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PHYSIOLOGIC OBSERVATIONS IN EXPERIMENTAL PULMONARY INSUFFICIENCY

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THE surgical attack upon isolated congenital stenosis of the pulmonary valve has been quite successful in relieving symptoms due to this disease. After transventricular valvulotomy (Brock technique),[1, 2] however, right ventricular pressures rarely return to normal and usually are in the range of 50 to 90 mm. Hg.[3-5] Pressure studies of a Brock procedure upon an eighteen-year-old boy are illustrated in Fig. 1. Preoperatively, the right ventricular pressure was 128/0. After the first cutting and dilatation of the valve with Potts' instruments the right ventricular pressure was 90/0. The valve was incised and dilated again with the instruments opened to the full extent but right ventricular systolic pressure could not be brought lower than 60 mm. Hg. The persisting systolic pressure gradient across the pulmonary valve probably is due to incomplete relief of stenosis, but the fact that the pulmonary artery and right ventricular pulse contours resemble each other during diastole after valvulotomy suggests that insufficiency of the valve may be contributing to the gradient (discussion follows). In an attempt to reduce right ventricular pressures to normal levels, Swan and associates[6-9] perform pulmonary valvulotomy under direct vision by temporarily occluding the circulation under hypothermia. By this means, they produce right ventricular pressures within the normal range.[6] The pulmonary valve apparently is rendered incompetent since diastolic murmurs are audible in most of the patients. Swan[6] and

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Blount,[7] and their associates, feel that this probably is of no significance since the patients get along well and the electrocardiographic right ventricular strain pattern decreases. Also, Swan has observed dogs with insufficiency of the pulmonary valve for several years and no evidence of cardiovascular disturbance has developed.

Hufnagel[10] produced some degree of insufficiency of the pulmonary valve in dogs by means of a technique to correct pulmonary stenosis. During the fourteen-week observation period, no evidence of dilatation or hypertrophy of the right ventricle developed.

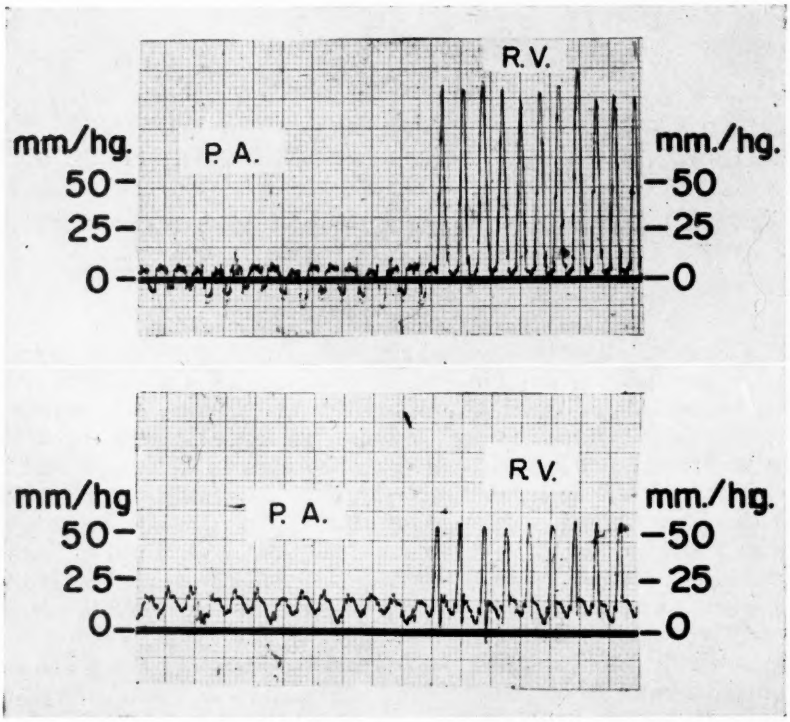


Fig. 1.—*A*, Preoperative cardiac catheterization record of an 18-year-old white man with isolated pulmonary stenosis. *B*, Postvalvulotomy record made at surgery. Note persisting systolic pressure gradient across pulmonary valve and similarity between P.A. and R.V. diastolic contours suggesting regurgitation.

After relief of the obstruction by the transventricular approach in tetralogy of Fallot, patients sometimes develop right heart failure, presumably due to pulmonary insufficiency if the obstruction is completely removed.[11, 12] In pure pulmonary stenosis, Brock[13] feels that the obstruction may be relieved too greatly, resulting in serious insufficiency of the valve. He feels, too, that the effects of pulmonary insufficiency may be as serious as those of aortic insufficiency.

In order to inquire further into this problem, the pulmonary valves were removed from a series of dogs. The animals survived with normal activity for months. The most striking abnormality, aside from murmurs, was a right ventricular systolic pressure that exceeded the pulmonary artery pressure by 2 to 40 mm. Hg. This was seen in dogs whose pulmonary orifices were smooth, of normal size, and lacking only the valves.

Some of the dogs were operated upon under hypothermia induced by immersion in an ice bath. When the rectal temperature reached 31° C., the dogs were prepared for surgery. Usually the temperatures dropped 3° to 5° C. further, so that during surgery temperatures were 26° to 28° C. Ex-

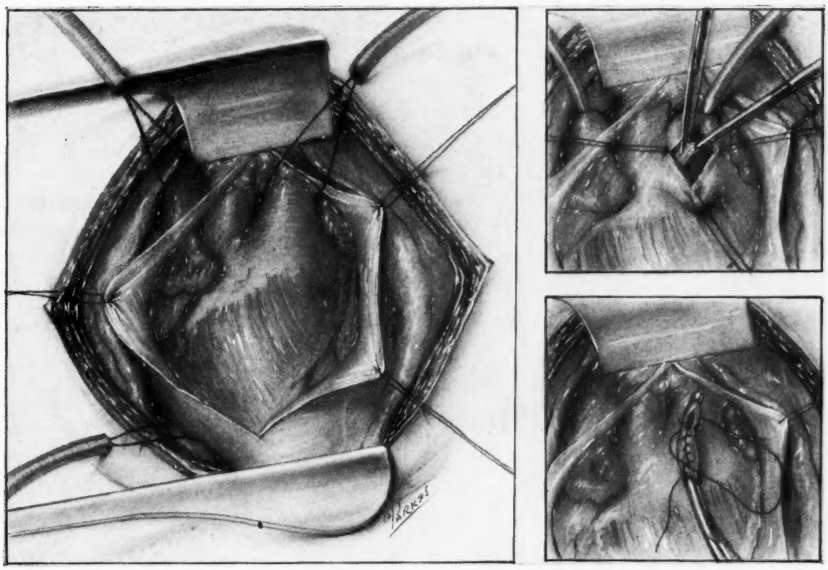


Fig. 2.—Operative exposure showing excision of pulmonary valves under direct vision during temporary occlusion of the circulation.

posure was obtained through transverse incisions which transected the sterum, both pleural cavities being entered in the third intercostal spaces anteriorly. The azygos vein was ligated. The venae cavae were occluded and after a few beats to empty the heart, the main pulmonary artery was occluded just proximal to its division. A 1.5 cm. to 2 cm. opening was made in the pulmonary artery just distal to the valve. Each valve cusp was grasped with forceps and excised (Fig. 2). A curved Potts' aortic clamp was placed across the opening in the pulmonary artery and occlusion ties were released so that circulation was re-established. Then a very careful repair of the pulmonary artery was done with No. 5-0 arterial suture. Usually circulation was occluded from 2 to 5 minutes. Ten dogs were operated upon in this manner. Most of them developed ventricular fibrillation and 5 were defibrillated electrically and survived.

Six dogs were operated upon under normothermic conditions. The operative procedure was the same except that occlusion of the circulation was limited to 2 minutes. If excision of the valve was not completed in this time, the pulmonary artery defect was clamped and circulation re-established for 5 or 10 minutes. While the circulation was occluded for a second 2-minute period, removal of the valves was completed. Five of the 6 dogs survived without developing ventricular fibrillation. Autopsy of the dogs that died in each group revealed complete removal of the valves with no obstruction of the pulmonary artery as a result of its repair.

Thus, 10 dogs survived total pulmonary valvulectomy and have been observed from one to fourteen months. The animals were turned into a dog run about two weeks postoperatively and have led normal lives. All have done well clinically and none has presented evidence of heart failure. The first 4 dogs of the series, under observation for eleven months or longer, have gone through apparently normal pregnancies.

In all dogs, phonocardiograms confirmed the presence of Grade III systolic and Grades I to II diastolic murmurs. We regard these murmurs as hemic rather than stenotic, because there was no anatomic stenosis. The presumably very large tidal ejection seems to cause a turbulence in the pulmonary trunk that is responsible not only for the murmur but also for the energy loss that is indicated by the pressure drop seen when the catheter tip leaves the ventricle and enters the artery.

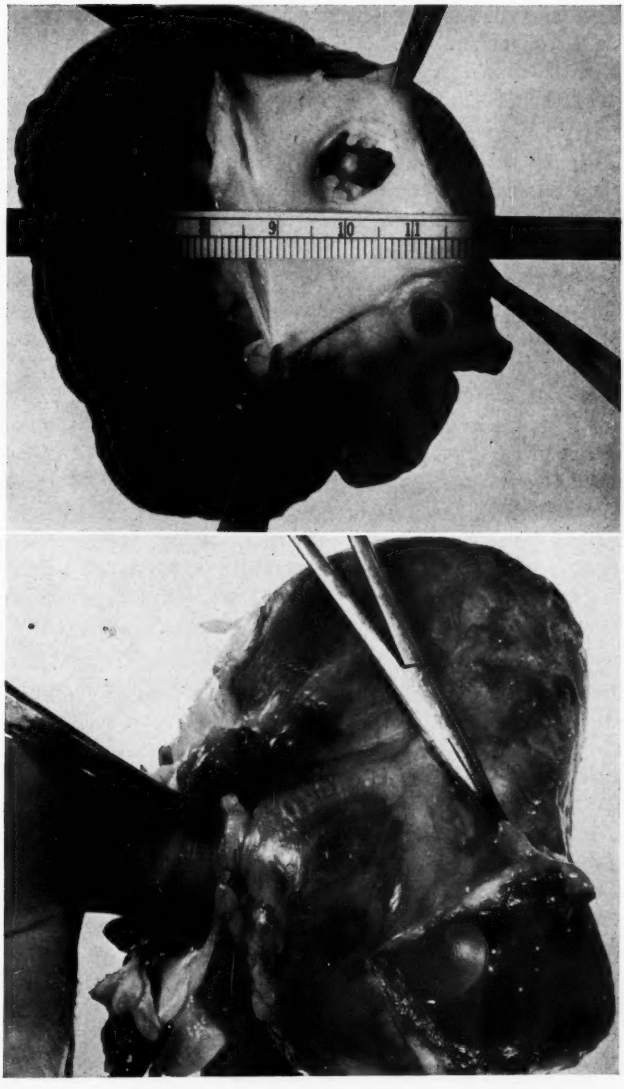
TABLE I. SUMMARY OF DATA ON DOGS WITH PULMONARY INSUFFICIENCY

DOG NUMBER	PERIOD OF OBSERVATION (MONTHS)	P.A. (MM. HG)	R.V. (MM. HG)	CARDIAC INDEX* FIRST AND LAST CATHETERIZATION	CARDIAC VOLUMES† PREOP. AND LAST X-RAY
HP-3	14	19/5	21/–3	2.2 – 2.7	542 – 463
HP-4	13½	11/3	23/1	5.6 – 4.6	403 – 359
HP-5	12½	30/2	70/3	2.9 – 1.8	440 – 310
HP-8	11	17/5	26/1	4.3 – 3.8	417 – 428
HP-10	1	28/–2	58/1	4.0	
NP-1	4	15/–1	20/–3	4.3	400 – 436
NP-2	4	14/1	33/2	4.0	455 – 533
NP-3	3½	25/3	33/4	3.8	458 – 342
NP-5	2½	29/3	37/3	4.3	365 – 623
NP-6	2½	21/1	30/1	4.5	453 – 593

*Liters per minute per square meter.
†Cubic centimeters per M.² body surface.

Cardiac catheterization was done at varying intervals after surgery. Some of the dogs have been catheterized three times while the more recent ones have been catheterized only once (Table I). In all cases, the right ventricular systolic pressure was greater than the pulmonary artery systolic pressure. This gradient averaged 15 mm. Hg for the 10 dogs with a range of 2 to 40 mm. Hg. One of these (HP-10) had a pulmonary artery systolic pressure of 28 mm. Hg and a right ventricular systolic pressure of 58 mm. Hg. This dog was sacrificed at one month and autopsy showed an unobstructed pulmonary orifice. The right ventricular wall was questionably thickened. The other dog with high right ventricular systolic pressure (HP-5) was followed

A.



B.

Fig. 3.—*A*, Photograph of dog heart (HP-5) showing patent pulmonary orifice. *B*, Index finger can be inserted through this area. Note slight hypertrophy of right ventricle.

for twelve and one-half months. At five months after operation, catheterization revealed a right ventricular systolic pressure of 61 mm. Hg and at ten months this pressure was 70 mm. Hg. Clinically the dog did well and presented no evidence of disturbance in circulation when sacrificed twelve and one-half months after surgery. Autopsy revealed no evidence of obstruction at the site of the pulmonary valves and the right ventricle was questionably hypertrophied (Fig. 3, *A* and *B*).

Pulmonary artery diastolic pressures were uniformly low, ranging from 0 to 5 mm. Hg. In no case was a normal diastolic pressure recorded. The pulmonary artery and right ventricular pulse contours resembled each other.

Right ventricular end-diastolic pressures were normal in all cases.

Cardiac outputs by the Fick method were essentially normal and no particular difference between the early and late cardiac indices was observed. In one case (HP-5), catheterization ten months after valvulectomy revealed a low output (cardiac index = 1.8), but no particular significance was attached to it because the dog presented no evidence of failure.

Measurement of heart volumes by x-ray,[14] as indicated in Table I, showed a significant increase in only 2 dogs. Both of them have been followed two and one-half months since surgery. One (NP-6) had a 30 per cent increase and the other (NP-5), a 70 per cent increase in heart volume. Both of them are doing well.

Electrocardiograms likewise were made preoperatively and at frequent intervals postoperatively. In no case was any evidence of right ventricular strain or hypertrophy present even though the 2 dogs autopsied showed suggestive hypertrophy.

DISCUSSION

The demonstration of a pressure gradient across the pulmonary orifice would ordinarily suggest a stenosis in this region. Autopsy of the two dogs which showed the largest RV-PA pressure gradient showed, however, that no such obstruction existed. The pressure drop must be explained as a result of something other than a stenosis. Kohout and Katz[15] recently reported a patient with pulmonary valvular insufficiency diagnosed by cardiac catheterization. Likewise, as in our dogs, the diastolic pressures in the pulmonary artery and right ventricle were similar and a systolic gradient was recorded across the valve area.

The amount of blood ejected during systole must be abnormally large because it includes the regurgitated blood as well as that coming through the tricuspid valve. Velocity of flow through the pulmonary artery would therefore be very great and, as indicated by the strong systolic murmur, the flow must be turbulent. The acceleration of the column of blood in the pulmonary artery to an unusually high velocity with its creation of turbulence in the stream may account for the pressure drop even in the absence of a demonstrable stenosis.

In a clinical study of atrial septal defects, Blount and co-workers[16] demonstrated a systolic pressure gradient across the pulmonary valve. He attributed

this gradient and the associated systolic murmur to functional pulmonary stenosis and turbulent flow in the pulmonary artery as a result of the high pulmonary blood flow. The murmur ceased and the pressure gradient disappeared when the abnormally high pulmonary flow was rendered normal by closing the defect.

If the diastolic pressure of the pulmonary artery were high like that of the systemic system, pulmonary insufficiency might lead to serious results. The pulmonary artery diastolic pressure is only 8 to 10 mm. Hg,[17] however, and after valvulectomy approaches zero. Dogs apparently tolerate the complete elimination of the pulmonary valves very well without heart failure.

This study, then, tends to support the clinical impression that in performing pulmonary valvulotomy for isolated pulmonary stenosis, some degree of insufficiency is acceptable in order to relieve stenosis completely. The fact that pulmonary regurgitation is compatible with health in normal hearts does not mean that it is necessarily so in the presence of a septal defect. Here the septal defect presents the diastolic right ventricle with so much blood that signs of strain often develop. Adding regurgitant blood to this might lead to trouble.

In cases of combined interventricular defect and pulmonary stenosis, the degree of stenosis in relation to the systemic peripheral resistance determines the proportion of blood ejected from the "common" ventricle which goes to the pulmonary circuit and to the systemic circuit. If the systemic resistance is too low in relation to the pulmonary stenotic resistance the ventricle discharges into the systemic circuit and the lungs get little or no blood, the right-to-left shunt is strengthened, and the patient becomes deeply cyanotic.[18] Conversely, the hazard of opening a pulmonary stenosis too widely by valvulotomy must be recognized. The pulmonary circuit, with low peripheral resistance, would then get more than its "share" of blood from the "common" ventricle. The shunt would be reversed and right ventricular strain might become dangerous. It is fortunate that the Blalock-Taussig short circuit is of such a size as to increase effectively pulmonary blood flow without lowering total resistance to pulmonary blood flow enough to bring about a dangerous imbalance between the flow to the pulmonary and systemic circuit.

SUMMARY

Clinical observations up to fourteen months, supported by data obtained from cardiac catheterization, electrocardiograms, and x-rays on 10 dogs, indicate that pulmonary insufficiency resulting from total pulmonary valvulectomy is tolerated remarkably well.

The regurgitating pulmonary blood augments the ejected volume so that there is a pressure gradient across the pulmonary orifice in the absence of stenosis.

The presence of septal defects can be expected to add to the right heart strain produced by regurgitation so that regurgitation may be tolerated better in the absence of septal defects than in their presence.

REFERENCES

1. Brock, R. C.: Congenital Pulmonary Stenosis, Am. J. Med. **12**: 706, 1952.
2. Brock, R. C.: Pulmonary Valvulotomy for the Relief of Congenital Pulmonic Stenosis, Brit. M. J. **1**: 1121-1126, 1948.
3. Lurie, P. R., and Shumaker, H. B.: Hemodynamic Effects of Valvulotomy in Pulmonic Stenosis, Circulation **8**: 345, 1953.
4. Bing, R. I., Reher, W., Sparks, J. E., Balboni, F. A., Vitali, A. G., and Hanlon, M.: Congenital Pulmonary Stenosis, J. A. M. A. **154**: 127, 1954.
5. Humphreys, G. H., II, Powers, S., Fitzpatrick, H., and Lanman, B. M.: Pulmonary Valvulotomy. Results of Operation in Twenty-Five Cases, Surgery **35**: 9, 1954.
6. Swan, H., Cleveland, H. C., Mueller, H., and Blount, S. G., Jr.: Pulmonic Valvular Stenosis, J. THORACIC SURG. **28**: 504, 1954.
7. Blount, S. G., McCord, M. C., Mueller, H., and Swan, H.: Isolated Valvular Pulmonic Stenosis. Clinical and Physiological Responses to Open Valvuloplasty, Circulation **10**: 161, 1954.
8. Swan, H., Zeavin, I., Holmes, J. H., and Montgomery, V.: Cessation of Circulation in General Hypothermia, Ann. Surg. **138**: 360, 1953.
9. Swan, H., and Zeavin, I.: Cessation of Circulation in General Hypothermia, Ann. Surg. **139**: 385, 1954.
10. Hufnagel, C.: A Method in the Correction of Pulmonary Stenosis. Surgical Forum, American College of Surgeons, Philadelphia, 1950, W. B. Saunders Co., p. 248.
11. Bailey, C. P.: Discussion of Brock, R. C.: The Results of Direct Operations on Pulmonary Stenosis in Fallot's Tetralogy. Presented before the Second World Congress of Cardiology, Washington, D. C., September, 1954.
12. Sell, C. G. R., Fowler, R. L., Sailores, E., Hyman, A. L., Levy, L., II, and Ordway, N. K.: Physiological Studies in Cases of Tetralogy of Fallot With Little or No Overriding of the Aorta. Presented before the Second World Congress of Cardiology, Washington, D. C., September, 1954.
13. Brock, R. C.: Panel Discussion on Congenital Heart Disease. Second World Congress of Cardiology, Washington, D. C., September, 1954.
14. Hamilton, W. F., Jr., Dow, P., and Hamilton, W. F.: Measurement of Volume of Dog's Heart by X-ray: Effect of Hemorrhage, of Epinephrine Infusion and of Buffer Nerve Section, Am. J. Physiol. **161**: 466, 1950.
15. Kohout, F. W., and Katz, L. N.: Pulmonic Valvular Regurgitation. Report of a Case with Catheterization Data, Am. Heart J. **49**: 637, 1955.
16. Blount, S. Gilbert, Jr., Swan, Henry, Gensini, Goffredo, and McCord, Malcolm C.: Atrial Septal Defect. Clinical and Physiological Response to Complete Closure in Five Patients, Circulation **9**: 801, 1954.
17. Hamilton, W. F.: The Physiology of the Pulmonary Circulation, J. Allergy **22**: 397, 1951.
18. Hamilton, W. F., Winslow, J. A., and Hamilton, W. F., Jr.: Notes on a Case of Congenital Heart Disease With Cyanotic Episodes, J. Clin. Invest. **29**: 20, 1950.

DISCUSSION

DR. FRANK GERBODE, San Francisco, Calif.—We have been interested in pulmonic stenosis because we have found a number of adults who have a very high right ventricular pressure and a very tight and hard valve. This valve can be cut and dilated in the way described by Brock and others, but the pressures do not always fall because the valve lacks elasticity. Some are almost cartilaginous in nature, and I believe they become that way because of the flow and turbulence caused by the stenosis itself. In other instances, the valve becomes more stenotic and hard because of fibrosis resulting from endocarditis. Obviously, some other means, as has been pointed out by Swan, must be called upon to cut these valves. We have done so in a number of these unusual instances by simply biting out a piece of valve with a small Brock punch valvulotome, it being necessary to do this in order to obtain a reduction in ventricular pressure.

These findings led to a study of pulmonic insufficiency in the laboratory. We have 10 animals under observation who have had total removal of the pulmonary valves. We too have found that pulmonary insufficiency is a relatively benign lesion in a normal animal. These animals run around the laboratory, act quite normally, and do not seem to get heart failure. However, we must, I believe, reserve our opinion on this matter of total excision of the pulmonic valve until we have made observations under stress and over a longer period of

time. Then we may find that the pulmonic valve is not quite so useless. On the other hand, mild pulmonary insufficiency may be well tolerated, and is probably much better than severe pulmonary stenosis.

DR. J. L. EHRENHAFT, Iowa City, Iowa.—I should like to make a short comment on the paper by Dr. Ellison. We have had the opportunity of observing and operating on a 14-year-old boy who was found to have a pure pulmonary valve insufficiency. There were no other cardiac valvular lesions present. This child was first seen at the University Hospitals during November, 1954, with the entrance complaint of having heart trouble for at least ten years. The parents had noticed some shortness of breath and some decreased exercise tolerance in the child since the age of 4. There had never been any cyanosis. Physical examination revealed a markedly enlarged heart with evidence of right ventricular overactivity. There was a loud systolic murmur and thrill over the pulmonic area and a loud diastolic murmur and thrill over the apex of the heart. The second pulmonic sound was not audible. Cardiac fluoroscopy revealed what was thought to be a pulmonic stenosis with tremendous poststenotic dilatation of the pulmonary artery and evidence of considerable right ventricular enlargement. An ECG revealed complete right bundle branch block and right ventricular enlargement. Angiocardiography was carried out and it demonstrated an aneurysmal dilatation of the pulmonary artery.

(slide) Cardiac catheterization revealed the following pertinent findings: Pressures in the superior vena cava, 80/4 mm.; right atrium, 6/2 mm. Hg; high right ventricle, 50/7 mm. Hg; low right ventricle, 50/7 mm. Hg; left proximal pulmonary artery, 27/10 mm. Hg; distal left pulmonary artery, 19/5 mm. Hg. The mean pulmonary artery pressure was in the proximity of 16 mm. Hg. The end diastolic pressure in the right ventricle was 7 mm. Hg. There was evidence of a pressure gradient of 20 mm. Hg across the pulmonic valve area. The stroke volume was 61 c.c. and the cardiac output was 4.6 L. per minute. The patient was subjected to operation on March 15, 1955. At that time a bilateral anterior thoracotomy was carried out and the heart was exposed. (slide) We did not use hypothermia but did occlude the superior and inferior vena cava and also the pulmonary artery. The pulmonic valve area was then visualized directly through an incision made in the markedly dilated pulmonary artery. The pulmonic leaflets were found to be completely destroyed.

We are reporting this case for two reasons: first, this is an unusual lesion which has been experimentally produced in animals but has not been reported to my knowledge in the human. Whether this lesion is secondary to rheumatic fever with isolated valvular involvement or due to destruction of the leaflets by an episode of bacterial endocarditis, I am unable to tell. Second, this child has been known to have this lesion for over ten years and has got along fairly well even though his exercise tolerance is reduced. We felt that this short presentation might have some bearing on the paper presented by Dr. Ellison.

EXPERIMENTAL TRANSPOSITION OF THE GREAT VESSELS: SOME FACTORS WHICH INCREASE THE FLOW THROUGH THE ATRIAL SEPTAL DEFECT

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A PARTIAL transposition was created in dogs by the following 3 procedures: (1) an artificial interatrial septal defect, (2) an anastomosis between the distal cut end of the right superior pulmonary vein and the proximal end of the azygos vein and, (3) an end-to-end anastomosis between the brachiocephalic artery and right pulmonary artery (Fig. 1). The purpose of this paper is to describe methods and results of increasing the right-to-left shunt through the atrial septal defect. The preparation just described is called the Type I since we have also created experimental transposition in another manner. A complete report on the Type I and Type II preparations will be presented in a separate communication.

METHODS AND RESULTS

Adult mongrel dogs were used. They were anesthetized with sodium pentobarbital given intravenously. Mean blood pressures were determined by inserting a needle which was connected to a mercury manometer into the femoral artery. During the catheterization of the heart, the blood pressure was recorded using strain gauges with a 2 channel Sanborn Visocardiette. Later studies were made with an optical recording oscillograph. The blood oxygen determinations were made with the Van Slyke-Neill apparatus.

The Type I Preparation.—After the right chest was opened through the bed of the fourth or fifth rib, an interatrial septal defect was made by the method of Swan and co-workers[8] in 12 of the 14 dogs included in this report. The divided distal end of the right superior pulmonary vein was anastomosed to the proximal end of the azygos vein. The proximal end of the pulmonary vein was closed by suture; the distal end of the azygos vein was ligated. The proximal end of the divided brachiocephalic (innominate) artery was then sutured end-to-end to the right pulmonary artery creating an artificial ductus arteriosus. The proximal end of the right pulmonary artery was ligated. By this arrangement, pulmonary vein blood from the right apical and cardiac lobes is diverted into the right auricle via the azygos vein and superior vena cava. There is an increase in flow in the right superior and inferior pulmonary veins due to the connection of the right pulmonary artery to the aorta via the brachiocephalic

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artery. The increase in flow into the left heart from the right inferior pulmonary vein tends to balance the additional flow into the right heart. To avoid repeated description of the operation this will be called the Type I preparation (Fig. 2).

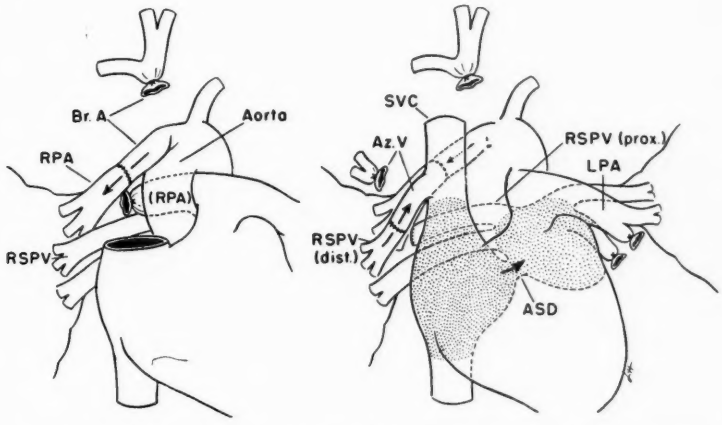

Fig. 1.—Diagram of the Type I preparation. *Br. A*: Brachiocephalic artery; *RPA*: Right pulmonary artery; *RSPV*: Right superior pulmonary vein; *SVC*: Superior vena cava; *Az.V*: Azygos vein; *ASD*: Atrial septal defect; *LPA*: Left pulmonary artery.

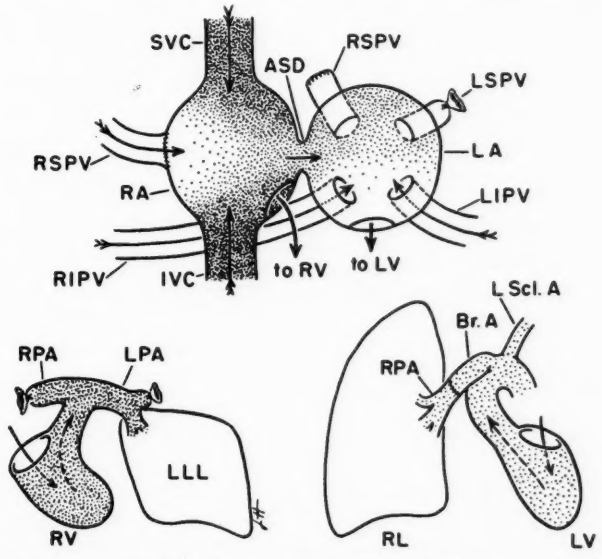

Fig. 2.—Diagram of the Type I preparation. Upper figure represents right and left auricles. The right superior pulmonary vein empties into the right atrium. An atrial septal defect is present.

The lower left figure represents the outflow of the right ventricle which is restricted to the left lower lobe. The end-to-end anastomosis of the brachiocephalic artery to the right pulmonary artery is illustrated in the lower right figure. Thus, the right lung receives its entire blood supply from the left ventricle and aorta and none from the right ventricle and pulmonary artery.

TABLE I. EFFECT ON ARTERIAL OXYGEN SATURATION OF TEMPORARY OCCLUSION OF THE LEFT PULMONARY ARTERY IN ANIMALS WITH THE TYPE I PREPARATION

	TIME	DOG 300 (2/6/53)	TIME	DOG 300 (2/13/53)	TIME	DOG 302 (2/4/53)	TIME	DOG 299 (2/17/53)	TIME	DOG 305 (3/13/53)	TIME	DOG 317 (5/16/53)
		(%)		(%)		(%)		(%)		(%)		(%)
Control arterial oxygen Breathing air		77		72		76		57		71		80
Control arterial oxygen Breathing oxygen												85
After occlusion of LPA	8½ min.	65	2 min. 20 sec.	66	55 sec.	55	2 min. 10 sec.	59	2 min.	49.8	1 min.	69
After release LPA			5 min.	71				80		80		
After reocclusion LPA	1½ min.	54			75 sec.	57						
After release LPA	3 min.	66										
After reocclusion LPA					2⅓ min.	59						

Dog 300 was under local anesthetic only, closed chest, breathing air. All others were under Pentothal sodium anesthesia, open chest, breathing 100 per cent oxygen with positive pressure.

Dog 302 had intact left lung, Dog 305 had left inferior lobectomy, Dogs 299, 300, and 317 had left superior lobectomy.

Time indicates period of occlusion of artery.

All 12 of the animals with the Type I preparation became cyanotic. The arterial oxygen saturation was measured in 6 dogs while breathing air without anesthesia. The average of 10 determinations was 70.5 per cent (range 57 to 80 per cent).

Three methods were used in the Type I preparations to increase the right-to-left shunt through the atrial septal defect. The first 2 methods were studied in acute experiments, the last was observed in chronic experiments. These were (1) left pneumonectomy, (2) temporary occlusion of the left pulmonary artery, and (3) excision of a lobe of the left lung.

The degree of cyanosis as measured by the arterial oxygen saturation is used as a rough estimate of the volume of the right-to-left shunt. Pulmonary hypertension consistently developed in the dogs which were maintained up to two years; however, presentation of those results will largely be deferred in order to simplify this presentation.

1. *Left Pneumonectomy (Experimental Pulmonary Atresia).*—The effects of left pneumonectomy were of interest, since ligation of the left pulmonary artery prevents any output from the right heart except through the interauricular septal defect. In 4 animals prepared with the Type I operation, pneumonectomy was performed. Two of these dogs had an interatrial septal defect and 2 did not. The 2 animals with an intact atrial septum promptly developed ventricular fibrillation when the left pulmonary artery was clamped. The 2 animals with an interatrial communication lived for two and one-half and six hours after left pneumonectomy. At autopsy, the right lung was congested and the bronchi and trachea contained white foam. It will be shown later that there is an immediate fall in the arterial oxygen content which must be a factor in the death of the animals. Right ventricular failure is another factor. When the right and left pulmonary arteries are completely occluded the atrial septal defect permits some emptying of the right ventricle. The survival for several hours of these 2 dogs is thus explained. If an interventricular septal defect was made one would anticipate that longer survival would be possible.[6] This condition corresponds to that seen clinically as *pulmonary atresia.*

2. *Temporary Occlusion of the Left Pulmonary Artery.*—The results in 5 experiments are summarized in Table I. Patency of the anastomoses and interatrial defect was demonstrated at autopsy in 3 dogs (300, 302, 305). Part of the record of Dog 317, including the autopsy description of the septal defect, is lost. Dog 299 is still alive. It may be assumed, however, that the septal defect was still patent in Dog 299 since ventricular fibrillation would have occurred immediately, as in the pneumonectomy experiments just described, when the septum was intact. The left pulmonary artery was occluded several times by traction on the ligature under local anesthesia with the chest closed in Dog 300. It is assumed that the occlusions were complete although not confirmed by direct vision. All the other occlusions (55 seconds to 2½ minutes) were by clamp under direct vision and it is certain that complete occlusion was obtained. The bronchial arteries are not occluded in these experiments. Dogs 299, 300, and 317 of this group had a left superior lobectomy,

Dog 305 had a left inferior lobectomy, and Dog 302 had an intact left lung. It was demonstrated in the experiments just described, in which pneumonectomy was performed, that occlusion of the left pulmonary artery, pulmonary veins, and bronchus may be tolerated for several hours if an interatrial septal defect is present. Arterial blood samples were drawn from the femoral artery in Dog 300 and from the aorta in the remainder.

A fall in the arterial oxygen saturation followed occlusion of the left pulmonary artery in all the animals. Presumably this is produced by an immediate increase (within 55 seconds) in the right-to-left shunt through the atrial septal defect. The greatest reduction occurred in Dog 305 which had the larger inferior lobe resected (24 per cent of lung tissue compared to the upper lobe which constitutes 16 per cent[5]).

Evidence that the fall in arterial oxygen content was not due to a general deterioration of the condition of the animal but to an increase in the right-to-left shunt through the patent atrial septum is derived from measurements of the systemic blood pressure before, during, and after the left pulmonary artery was occluded (Table II). A rise in blood pressure was noted in the 2

TABLE II. EFFECT ON ARTERIAL BLOOD PRESSURE OF TEMPORARY OCCLUSION OF THE LEFT PULMONARY ARTERY IN ANIMALS WITH THE TYPE I PREPARATION

DOG 302 (2/4/53) PENTOBARBITAL ANESTHESIA, OPEN CHEST. 100% 0_2 POSITIVE PRESSURE		DOG 300 (2/6/53) LOCAL ANESTHESIA, CLOSED CHEST. BREATHING AIR	
TIME (MINUTES) *Control*	MEAN BP AORTA (MM. Hg) *64*	TIME (MINUTES) *Control*	BP FEMORAL ARTERY (MM. Hg) *120/80*
0	LPA clamped	0	LPA occluded
½	64	4	120/80
1½	80	6	105/80
2	100	8½	135/80
4	LPA released	8½	LPA released
4	120	11	115/85
6	120	15	110/85
7	102	15	LPA reoccluded
9	100	16½	160/85
		18	LPA released
		18½	150/90

animals in which these studies were made. One was under sodium pentobarbital anesthesia, with 100 per cent oxygen administered through an endotracheal tube with open thoracotomy; the other experiment was under local anesthesia with a closed chest, the animal breathing air. An early increase in the systemic blood pressure could be explained by the acute effect of anoxia producing vasoconstriction. Eventually right ventricular failure, due to inability to empty adequately, would result in a fall in cardiac output and blood pressure. This factor would then contribute to the hypoxemia. Left ventricular failure might also occur late but would not account for the rapid fall in arterial oxygenation. It is doubtful whether the systemic blood pressure would have remained normal had the experiments continued for several hours.

3. *Lobectomy.*—In 6 animals, a lobectomy of the left lung was performed. The inferior lobe (larger) was resected in 2 and the superior lobe in 4 animals.

These procedures were well tolerated. The arterial oxygen saturation was determined in 1 dog of this group before and after left superior lobectomy. In Dog 300, there was no apparent effect (75 per cent before lobectomy, 77 and 72 per cent two months after lobectomy). The relation of the reduction in the vascular bed by left lobectomy to the development of cyanosis or pulmonary hypertension will be mentioned later under "Discussion." Experimental data to show the effect of reduction of the vascular bed are included in the preceding section concerning the effects of temporary occlusion of the left pulmonary artery.

DISCUSSION

Survival of dogs with experimental pulmonary atresia (right lung supplied with blood from aorta via right pulmonary artery and left pulmonary artery divided at a second stage) depends on the right ventricle being able to empty itself via an atrial or ventricular septal defect, or both. A reverse flow ductus is another possible way for the right ventricle to empty. In our experiments, only an atrial septal defect was created.

The concept of experimentally forcing a greater flow from right to left through an atrial septal defect by increasing the resistance to the outflow of the right ventricle has a clinical analogy. In clinical cases of pulmonic stenosis associated with a patent foramen ovale or an atrial septal defect, severe degrees of cyanosis have been observed.[3, 4] After relief of the pulmonic stenosis, the cyanosis is reduced. In both experimental and clinical examples, the preoperative pressure in the right ventricle and right atrium is elevated and increases the right-to-left shunt.

Temporary occlusion of the left pulmonary artery in the Type I preparation was followed by an immediate and pronounced fall in arterial oxygen saturation. This reproduces the condition long observed clinically of cyanosis produced by crying, pneumonitis, etc., which raise the pressure on the right side of the heart or in the pulmonary artery. This was emphasized by Maude Abbott[1] when she classified patent ductus and septal defects as "cyanosis tardive."

A more chronic type of cyanosis occurs clinically with large atrial septal defects or those with right ventricular failure and pulmonary hypertension. Chronic cyanosis also occurs when atrial septal defects are associated with other defects such as pulmonic stenosis or transposition of the great vessels. Excision of a lobe of the left lung in the Type I preparation was an attempt to increase the right ventricular and pulmonary artery pressures thus producing a deeper degree of cyanosis. Ferguson and Varco[5] have recently shown experimentally in puppies that subtotal pulmonary resection is followed by pulmonary hypertension and endothelial proliferative changes in the small pulmonary arteries. There is abundant clinical evidence that lung destruction by chronic fibrosis and emphysema, for example, is followed by pulmonary hypertension and cor pulmonale.

The relation of the atrial septal defect to the surgical relief of complete transposition of the great vessels has been pointed out by Blalock and Hanlon.[2]

They found that the creation of a large atrial septal defect, together with an anastomosis between the subclavian and pulmonary arteries, was followed by the greatest improvement in their patients. They also noted in their series that a patient who happened to have a mild pulmonic stenosis and therefore theoretically a greater right-to-left shunt as well as transposition of the great vessels had the most striking improvement.

SUMMARY

1. Partial transposition of the great vessels has been created by end-to-end anastomosis between the right superior pulmonary vein and the azygos vein and between the brachiocephalic artery and right pulmonary artery. In addition, an atrial septal defect is created.

2. The right-to-left shunt was increased in this preparation by three methods: (1) left pneumonectomy (corresponds to experimental pulmonary atresia), (2) temporary occlusion of the left pulmonary artery (corresponds to Abbott's "cyanosis tardive"), and (3) excision of a lobe of the left lung (corresponds to pulmonary destruction and cor pulmonale).

REFERENCES

1. Abbott, Maude E.: Osler's Modern Medicine, Philadelphia, 1927, Lea & Febiger, Vol. 4, p. 653.
2. Blalock, A., and Hanlon, C. R.: The Surgical Treatment of Complete Transposition of the Aorta and Pulmonary Artery, Surg., Gynec. & Obst. 90: 1, 1950.
3. Blalock, A., and Kieffer, R. F., Jr.: Valvulotomy for the Relief of Congenital Valvular Pulmonic Stenosis With Intact Ventricular Septum, Ann. Surg. 132: 496, 1950.
4. Engle, M. A., and Taussig, H. B.: Pulmonic Stenosis With Patent Foramen Ovale, Circulation 2: 481, 1950.
5. Ferguson, D. J., and Varco, R. L.: The Relation of Blood Pressure and Flow to the Development and Regression of Experimentally Induced Pulmonary Arteriosclerosis, Circ. Research 3: 152, 1955.
6. Hanlon, C. R., and Blalock, A.: Complete Transposition of the Aorta and Pulmonary Artery, Ann. Surg. 127: 385, 1948.
7. Martin, W. B., and Essex, H. B.: Experimental Production and Closure of Atrial Septal Defects, With Observation of Physiologic Effects, Surgery 30: 283, 1951.
8. Swan, H., Maresh, G., Johnson, M. E., and Warner, G.: The Experimental Creation and Closure of Auricular Septal Defects, J. THORACIC SURG. 20: 542, 1950.

DIRECT VISION INTRACARDIAC SURGERY BY MEANS OF A RESERVOIR OF "ARTERIALIZED VENOUS" BLOOD

Description of a Simple Method and Report of the First Clinical Case

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OVER the past year at the University of Minnesota Hospitals controlled cross circulation[1] has been used successfully in the curative management of over 40 patients with interventricular, atrioventricularis communis, or tetralogy of Fallot defects.[3] The clinical experience with this method of performing open intracardiac surgery for prolonged periods has emphasized two important facts relevant to simplification in the method of perfusion during the period of total cardiac by-pass.

The first of these has been confirmation of the practicability of the low flow principle.[2] The perfusion rates used in these patients represented only a small fraction of their basal cardiac output and ranged from 25 to 40 c.c. per kilogram body weight per minute. Notwithstanding these low flows during the period of by-pass, there was no central nervous system damage evident in any of these patients nor did any demonstrate impairment of liver or kidney function postoperatively. Also, the cardiac action in these cases further attested to the feasibility of these flows in that even in the presence of a generous ventricular cardiotomy and surgical manipulation the myocardium consistently maintained good color and contractions. Ventricular fibrillation occurred only once in this entire series. This patient was promptly defibrillated after closure of the cardiotomy and recovered without sequelae.

Second, the experience gained in corrective surgical treatment of these types of congenital heart defects indicated that, with direct vision, long periods of intracardiac operating time were not usually necessary in order to effect these surgical repairs. Well over three-fourths of these cases took less than 15 minutes of perfusion time to accomplish the intended intracardiac reparative procedure and all but 2 of the remaining patients required less than 20 minutes of perfusion. These two exceptions occurred earlier in the series when intracardiac experience was smaller.

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The result of these observations made during the use of controlled cross circulation in the clinical operating room was the realization that, especially in the infants and small children, the total amount of blood exchanged during the perfusion period was relatively small and conceivably could be provided by a reservoir instead of a donor. This suggested further investigation of a reservoir method of perfusion that we had employed to a limited extent in the laboratory some 12 to 14 months before. By the use of this method in conjunction with the concept of reduced flow and the mechanical principles of controlled cross circulation (which insured a quantitative balance between the arterial perfusion and the venous withdrawal from the recipient) it was possible to occlude the cardiac inflow and provide ample opportunity to operate within the chambers of a dry heart and at the same time to simplify the procedure.

METHOD

The arrangement of the extracorporeal circuits is expressed diagrammatically in Fig. 1. In general, the setup is similar to that employed in the controlled cross circulation technique[1] with the exception that the donor is replaced by an arterial *(A, 1* and *2)* and a venous *(V, 2* and *3)* reservoir. These are merely siliconized Baxter Plasma Vac bottles (500 c.c. or 1,000 c.c.) from which the vacuum has been exhausted. Bottles *A, 1* and *2* contain arterial blood that has been collected previously in the manner described below. *V, 2* and *3* are empty at the outset and are provided to collect the venous blood withdrawn from the patient during the perfusion.

It will be noted that the cannulations in the patient and their attachments to the pumping device are identical with those already described for use in controlled cross circulation. These may be seen in the diagram of the heart where it will be noted that a cannula has been inserted into and passed down the right subclavian artery until the tip lies in the ascending aorta. It is through this cannula that arterial blood from the reservoir enters the patient's arterial system. Also, it will be noted that a Rumel tourniquet has been placed about the base of the aorta in order to provide a means for intermittent occlusion if the aortic valves are found incompetent when the heart is open. In addition, in this same diagram, it will be noted that cannulae have been passed through the right auricular wall and into the superior and inferior venae cavae, respectively. These are secured by purse-string sutures at their points of entrance into the auricle and are joined at their distal ends to a "Y" adaptor. During the period of cardiac by-pass, when the umbilical tapes about the superior and inferior venae cavae are drawn up about their respective cannulae, venous blood will be channeled from the patient's caval system into these cannulae and none will enter the heart.

The extracorporeal circuits are assembled by first attaching two bottles of arterial blood to an ordinary blood filter* *(A, 3)* and allowing blood to fill the filter chamber while the latter is held in the inverted position so that all air will be displaced. The outflow of the filter is then attached to the afferent plastic tube of the arterial pump *(A, 4)*. The pump is then turned on momentarily so that the entire arterial circuit is filled with blood. When this is done and the venous circuit is assembled as diagrammed, the appropriate connections can be made to the patient's cannulae and perfusion begun. In practice, the arterial pump draws blood from only one bottle at a time. When the first bottle is nearly empty, as, for instance, *A, 2* in the diagram, its outflow is clamped, that of *A, 1* is released and perfusion goes on uninterrupted while *A, 2* is being replaced by a full bottle of arterial blood. This procedure is repeated until the

*Baxter Laboratories, Morton Grove, Illinois, Filter R-19.

pump is stopped. Similarly, on the venous side when *V, 2* is full the venous outflow from the pump is diverted into *V, 3* while *V, 2* is replaced by another empty bottle.

The output of each pump is preset at equal rates before the pump is brought to the operating room. It is desirable to check this calibration just prior to the onset of perfusion. This is done under sterile conditions using a 5 per cent glucose solution in order to avoid the waste of blood.

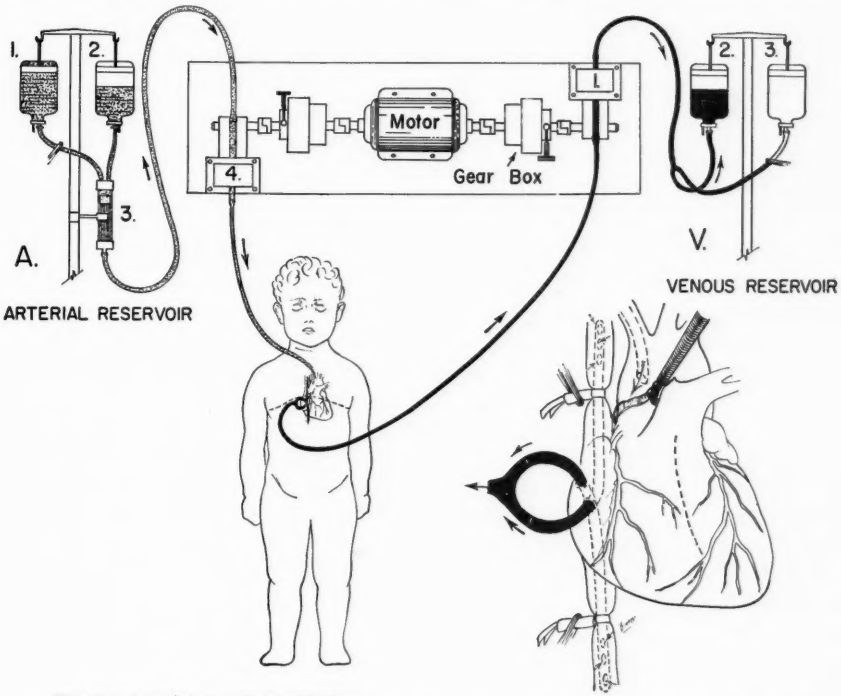

Fig. 1.—Arterial reservoir perfusion.

A, The arterial reservoir showing the bottles of arterial blood (*1, 2*), blood filter (*3*), and arterial pump (*4*).

V, The venous portion of the perfusion circuit with the venous pump (*1*) and the empty bottles (*2, 3*) for the collection of the venous blood withdrawn from the patient's caval system.

At the top center is the pumping assembly which consists of separate pumps at each end driven by a single motor (center) and separate speed changers which are interposed between the motor and each pump head. The patient's relationship to the system is depicted in the center of the diagram. In the lower right is a close-up view of the cannulations about the heart. The arterial catheter can be seen with its tip lying just within the ascending aorta and the venous catheters and their respective occluding tapes can be seen in their proper positions in the venae cavae. A Rumel tourniquet has been placed about the base of the aorta in order to ensure a dry intracardiac field by intermittent occlusion of this region if the aortic valves are found incompetent when the heart is opened. This latter maneuver also selectively controls the flow in the coronary arterial system.

COLLECTION OF ARTERIAL BLOOD

This method of reservoir perfusion requires the procurement, collection, and storage of arterial blood with which to supply the arterial reservoir.

Anticoagulants and Storage.—Heparin has been used as the anticoagulant instead of A.C.D.* solution because of the marked reduction in blood pH caused

*Acid citrate dextrose.

by the citric acid and the toxicity of citrated blood on the myocardium. At the outset, we adopted the amount of heparin frequently used for exchange transfusions which ranged between 50 and 75 mg. per 500 c.c. of blood. This amount was quite satisfactory with respect to the preservation of the blood, but it produced abnormal postoperative hemorrhage in all of the animals.[4] By further study, the optimum amount of heparin to prevent in vitro clotting without abnormal postoperative hemorrhage in the animal was found to be 15 to 17 mg. per 500 c.c. of blood, with marginal limits at 12.5 and 20 mg. In order to enhance mixing, this amount of heparin was added to 50 c.c. of 5 per cent glucose solution and was instilled in each 500 c.c. siliconized bottle after removal of the vacuum. This amount provided adequate protection against clotting with relatively good preservation of the cellular components over a fairly long period of time and under varying conditions.

Ordinarily, the arterial blood utilized for the perfusions has been collected within 2 to 4 hours of the time it was used. During this intervening period, it has been kept in a water bath at 38° C. to obviate the previously described deleterious effects on the heart of perfusion with cold blood.[3] However, it would seem reasonable to anticipate that storage for a prolonged period might be desired. In this event, the comparison of the depreciation noted when blood was maintained at room temperature (Table I) to that which occurred when stored at 4° C. (Table II) indicated that refrigeration was preferable for longer periods of storage. Inspection of these data revealed a rapid deterioration of the pH and oxygen content in the blood at room temperature over a few hours whereas the cold blood was stable, particularly with reference to the pH, for a much longer time. The platelets on the other hand tended to show a steady decline regardless of the mode of storage. The apparent inconsistency of the last count in Table II can be ascribed only to the wide margin of error inherent in platelet counts.

TABLE I. ARTERIAL BLOOD (HUMAN) COLLECTED IN HEPARIN* AND STORED AT ROOM TEMPERATURE

STORAGE TIME	pH	O_2 (VOL. %)	CO_2 (VOL. %)	HEMOGLOBIN	PLATELETS
Control	7.52	17.60	50.30	12.7	584,000
1 hour	7.51	18.00	43.98		410,000
2 hours	7.49	17.21	44.46		382,000
3 hours	7.48	16.6	43.10	12.0	298,000
4 hours	7.41	16.0	42.02		320,000
5 hours	7.35	16.88	42.67		344,000

*15 mg. heparin in 50 c.c. 5 per cent glucose per 500 c.c. blood.

TABLE II. ARTERIAL BLOOD (HUMAN) COLLECTED IN HEPARIN* AND STORED AT 4° C. TEMPERATURE

STORAGE TIME	pH	O_2 (VOL. %)	CO_2 (VOL. %)	HEMOGLOBIN	MEAN PLATELETS COUNT
Control	7.52	20.12	47.53	14.2	368,000
1 day	7.46	20.67	48.67	14.5	102,000
2 days	7.40	20.27	48.25	14.4	63,000
4 days	7.28	20.45	47.36	14.1	65,000
6 days	7.17	20.15	46.50	15	125,000

*20 mg. heparin in 25 c.c. 5 per cent glucose per 500 c.c. blood.

Single Donor (Donor Oxygenation).—Arterial blood can be obtained satisfactorily by using a donor individual (of compatible blood type) as an oxygenator entirely independent of the perfusion setup. This was done by cannulating the femoral artery and saphenous vein under local anesthesia and collecting the arterial blood into a bottle containing the heparin glucose solution previously described. This was done with the bottle in the inverted position to insure adequate mixing with the anticoagulant and by gravity plus very gentle suction to avoid excessive turbulence. Synchronous with this withdrawal of the arterial blood, the donor was infused with an equal quantity of ordinary citrated bank blood via the cannula in the saphenous vein. This infusion was given at a rate equal to that of the withdrawal of blood from the arterial system in order to avoid any alteration of the donor's blood volume. Rapid exchange can be done without untoward systemic reaction provided the replacement is infused into the same region of the circulatory system from which the arterial blood is withdrawn (i.e., infusion into the inferior caval venous system if collection is from the lower half of the arterial circuit). The changes produced in the donor by this procedure are minimal as evidenced by the experimental studies[4] in which animals processed volumes greater than their own volume with only a small drop in hemoglobin content (1.0 Gm.) and a 25 to 30 per cent drop in their platelet counts. Return to normal values occurred within 24 to 36 hours.

This procedure for procurement of arterial blood has been used on 2 average-sized human donors. In each instance, from 3,500 to 4,000 c.c. of arterial blood was obtained from a femoral artery cannula inserted under local procaine anesthesia and a like amount of venous blood was infused through a similar cannula in the vein. These procedures were carried out over a period of 60 to 90 minutes, each donor was up and about within a short period, and neither presented any early or late complications.

Multiple Donors (Arterialized Venous Blood).—The method just described seemed cumbersome when our attention was drawn to the early work of Meakins and Davies,[5, 6] Drucker and Cullen,[7] and others,[8, 9] who observed that blood samples from the antecubital vein of an arm that had previously been warmed in a water bath at 45 to 47° C. for 15 minutes was strikingly similar to arterial blood.

In order to demonstrate this effect of local heat, venous blood samples were drawn simultaneously from the normothermic and the warmed arms of the same individual. The results of this study are given in Table III. Particularly impressive is the relative difference in pH and O_2 contents of the samples drawn in this fashion which closely parallel the differences described by Meakins.[6]

TABLE III. COMPARATIVE STUDY OF pH, O_2, AND CO_2 CONTENT OF VENOUS AND "ARTERIALIZED VENOUS" BLOODS DRAWN SIMULTANEOUSLY* FROM THE SAME INDIVIDUAL

BLOOD SAMPLES	pH	O_2 (VOL. %)	O_2 (% SATURATION)	CO_2 (VOL. %)	HEMOGLOBIN
Normal vein	7.35	12.96	57.2	63.89	13.4
Arterialized vein	7.42	21.35	94.2	52.15	13.4

*Samples drawn simultaneously from the right and left antecubital veins of the same person (M.G.A.). The left arm only had been heated in water bath at 45° C. for 15 minutes.

The mechanism by which this arterialization of venous blood occurs is the result of a local increase in rate of circulation[10] and the opening up of multiple small arteriovenous anastomoses in response to the increased temperature of the extremity.[11, 12] Furthermore, the value of these physiologic observations was increased when through experimentation it became evident that by continued heating of the arm the resultant arterial character of the venous blood could be maintained for protracted periods. By adapting these observations to our needs, a simple method of obtaining large volumes of arterialized blood evolved as follows: The arm of the prospective donor was immersed in a water bath at 45° C. for 15 to 20 minutes. To facilitate collection he was then asked to recline on a cot and the water bath was replaced by hot packs maintained at approximately 45° C. in order to maintain the local hyperthermia during the collection. Then, by means of a simple venepuncture in the antecubital fossa, 500 c.c. of arterialized venous blood was collected by gravity from each donor in the fashion previously described.

The volume of blood collected in preparation for a given operation was determined by the flow rate desired, the size of the patient, and the estimated length of perfusion needed. In the clinical case described below approximately 4,000 c.c. of arterialized venous blood was drawn in the above fashion.

CASE REPORT

L. O., U. H. No. 880964, a 6-month-old male infant, entered the hospital on March 8, 1955. His parents stated he was well until the age of 3 months when his weight gain fell off and a heart murmur was heard on physical examination. In addition he had a history of frequent respiratory infections and dyspnea during the interval between 3 and 6 months of age.

Physical Examination.—There was a definite systolic thrill palpable over the precordium, being most noticeable in the left third and fourth interspaces, and a Grade IV harsh systolic murmur was also best heard in this area. The pulmonic second sound was greater than the aortic second sound. The liver margin was palpable 3 cm. below the right costal margin. A right cardiomegaly and increased pulmonary vascular markings were noted on x-ray examination of the chest. Cardiac catheterization revealed a jump in the blood oxygen saturation of 22 per cent between the right atrium (62 per cent) and the right ventricle (84 per cent) and a pressure of 45/0 mm. Hg in the outflow tract of the right ventricle. These findings indicated an interventricular septal defect.

On March 29, 1955, the patient was operated upon and a high interventricular septal defect, approximately 1 cm. in diameter, was found and closed under direct vision by using the multiple interrupted suture technique.[13] During the 14 minutes and 45 seconds of total cardiac by-pass necessary to close this defect, the patient (weight 4.8 kilograms) was perfused at a rate of 150 c.c. per minute (31 c.c./kg./min.) by means of the described reservoir method. Arterialized venous blood for the arterial reservoir had been drawn a few hours preoperatively in the blood bank from 8 compatible donors whose arms had been heated to 45° C. for 15 minutes prior to collection. A total of 3,950 c.c. was drawn in this fashion and available for perfusion; however, only 2,225 c.c. were needed. His postoperative laboratory studies revealed a platelet count of 189,000 and normal bleeding and clotting times with a hemoglobin of 13.4 Gm. The loss from the chest catheters yielded only 110 c.c. of serosanguineous fluid during the first 48 hours. Postoperatively, the patient's convalescence was smooth and uneventful. The cardiac murmur and thrill have disappeared.

DISCUSSION

The described reservoir method of perfussion provides a satisfactory means by which the circulation of oxygenated blood to the vital centers may be maintained during the period of cardiac by-pass in a patient undergoing an intracardiac operative procedure. It has the advantage of being simple in concept and in application. The materials used are readily available to any hospital, no elaborate devices are needed, and the operation and maintenance of the equipment not dependent upon the availability of highly trained technical personnel. The sole limiting feature of the method is the amount of blood available. However, in view of the relatively short periods of perfusion time needed to perform intracardiac operations in selected patients, the importance of this objection becomes more theoretical than practical. This is particularly true in regard to the application of the method to infants and small children who comprise many of the patients requiring intracardiac procedures. In such individuals, as in the case reported, the amount of blood used for the entire perfusion was less than that required to prime many of the existing extracorporeal perfusion systems.

The feasibility of this method is further enhanced by utilizing the physiologic phenomenon of the arterialization of venous blood by local heat during the collection of the reservoir blood. In this fashion large volumes of blood with the chemical characteristics of arterial blood can be obtained by the usual phlebotomy in the blood bank. Arterial blood obtained in this fashion and delivered by the reservoir system provides the patient with the same physiologic benefits of blood adjusted by the homeostatic mechanisms of a normal donor as in controlled cross circulation[1, 4, 14-16] but without any risk to the donor.

SUMMARY

1. An uncomplicated method that provides an adequate circulation of oxygenated blood to the vital organs of the body during the period of cardiac by-pass in patients undergoing operations within the open chambers of the heart is described. This is accomplished by a simple pump which simultaneously delivers blood from an arterial reservoir to the arterial system of the patient and withdraws an equal volume of venous blood from the vena caval system.

2. A simple method of obtaining blood possessing arterial chemical characteristics with which to supply the arterial reservoir is discussed. This can be done by utilizing the physiologic observation that blood drawn from a vein of an extremity that has been subjected to an external heat of 45 to 47° C. for 15 to 20 minutes is arterialized. Thus, by preheating the arms of blood bank donors, relatively large quantities of arterialized venous blood can be collected with ease.

3. The first patient in which a high interventricular septal defect was repaired under direct vision using the described reservoir method of perfusion and arterialized venous blood is reported. This infant has made an uncomplicated recovery.

4. The advantages of the method and the feasibility of its clinical application, especially to small patients, are discussed in detail.

To date, this method of reservoir perfusion for total by-pass of the heart and lungs for as long as forty-five minutes has been utilized successfully in 5 patients with various types of intracardiac defects.

The patient herein reported was seen recently in the Outpatient Department on Oct. 10, 1955. He had gained 7 pounds 10 ounces over his preoperative weight, was active without limitation, has been free of intercurrent illness, and was developing normally in marked contrast to his preoperative status. On physical examination no thrills or murmurs were present and on x-ray examination of the chest a regression of the pulmonary vascular pattern to normal limits was evident.

REFERENCES

1. Warden, H. E., Cohen, M., Read, R. C., and Lillehei, C. W.: Controlled Cross Circulation for Open Intracardiac Surgery, J. THORACIC SURG. **28**: 331, 1954.
2. Cohen, M., and Lillehei, C. W.: A Quantitative Study of the Azygos Factor During Vena Caval Occlusion in the Dog, Surg., Gynec. & Obst. **98**: 225, 1954.
3. Lillehei, C. W.: Controlled Cross Circulation for Direct Vision Intracardiac Surgery, Postgrad. Med. **17**: 388-396, 1955.
4. Warden, H. E., DeWall, R. A., Read, R. C., Aust, J. B., Cohen, M., Varco, R. L., and Lillehei, C. W.: Total Cardiac By-Pass Utilizing Continuous Perfusion From a Reservoir of Oxygenated Blood, Proc. Soc. Exper. Biol. & Med. vol. 90, November, 1955.
5. Meakins, J., and Davies, H. W.: Observations on the Gases in Human Arterial and Venous Blood, J. Path. & Bact. **23**: 451, 1919-20.
6. Meakins, J., Dautrebande, L., and Fetter, W. J.: The Influences of Circulatory Disturbances on the Gaseous Exchange of Blood. (IV. The Blood Gases and Circulation Rate in Cases of Mitral Stenosis), Heart **10**: 153, 1923.
7. Drucker, P., and Cullen, G. E.: A Simple Method for Obtaining Cutaneous (Capillary) Blood From Infants and Adults for Colorimetric pH Determinations, J. Biol. Chem. **64**: 221, 1925.
8. Davenport, H. W.: The ABC of Acid Base Chemistry, ed. 3, Chicago, Ill., 1950, University of Chicago Press, p. 80.
9. Clark, W. M.: Topics in Physical Chemistry, ed. 2, Baltimore, 1952, The Williams & Wilkins Company.
10. Dautrebande, L., Davies, H. W., and Meakins, J.: The Influences of Circulatory Disturbance on the Gaseous Exchange of the Blood. (III. An Experimental Study of Circulatory Stasis), Heart **10**: 133, 1923.
11. Maximow, A. A., and Bloom, W.: A Textbook of Histology, Philadelphia, 1942, W. B. Saunders Company, p. 255.
12. Popoff, N. W.: The Digital Vascular System, Arch. Path. **18**: 295, 1934.
13. Lillehei, C. W., Cohen, M., Warden, H. E., and Varco, R. L.: The Direct Vision Intracardiac Correction of Congenital Anomalies by Controlled Cross Circulation (Results in 32 Patients), Presented at the Meeting of the Society of University Surgeons, Houston, February, 1955. Surgery **38**: 11-29, 1955.
14. Warden, H. E., Cohen, M., DeWall, R. A., Schultz, R. A., Buckley, J. J., Read, R. C., and Lillehei, C. W.: Experimental Closure of Interventricular Septal Defects and Further Physiologic Studies on Controlled Cross Circulation, Surgical Forum, Philadephia, 1954, W. B. Saunders Company, pp. 22-28.
15. Lillehei, C. W., Cohen, M., Warden, H. E., Read, R. C., DeWall, R. A., Aust, J. A., and Varco, R. L.: Direct Vision Intracardiac Surgery by Means of Controlled Cross Circulation or Continuous Arterial Reservoir Perfusion. Symposium on Cardiovascular Surgery, Philadelphia, 1955, W. B. Saunders Company, pp. 371-392.
16. Lillehei, C. W., Cohen, M., Warden, H. E., Read, R. C., DeWall, R. A., Aust, J. A., and Varco, R. L.: Direct Vision Intracardiac Surgical Correction of the Tetralogy of Fallot, Pentalogy of Fallot, and Pulmonary Atresia Defects. Report of First Ten Cases, Ann. Surg. **142**: 418-445, 1955.

DISCUSSION

DR. RAYMOND C. READ, Minneapolis, Minn.—I should like briefly to supplement Dr. Warden's presentation by describing the chemical characteristics of arterialized venous blood taken from the antecubital vein following warming of the hand and forearm.

(slide) These are samples drawn from individual donors whose blood was used in a human case. One can see that the oxygenation, CO_2 concentration, and pH are essentially the same as found in the artery. The blood was drawn after immersing the donor's arm in water at 45° C. for 10 minutes.

(slide) We have recently studied the effect of ventilating the donor with oxygen during the drawing of the blood. This maneuver alone causes an average rise of 2 volumes per cent in the venous oxygen concentration primarily because of the increase in dissolved oxygen due to the raised partial pressure. Supplementation with warming of the arm resulted in a mean oxygen saturation in these 5 individuals of somewhere near 98 per cent, assuming all the oxygen to be carried in the hemoglobin. This slide also shows the differences between the control venous pH and CO_2 concentration, and those values obtained after warming and breathing of oxygen.

The last slide illustrates the storage characteristics of arterialized venous blood collected into heparinized glucose solution. As is to be expected, this blood rapidly deteriorates at room temperature. However, at 4° C., its arterial nature persists for at least two or three weeks. The platelet count drops consistently at a rate of approximately 50 per cent the first day in the icebox. For this reason, in all its human applications so far, arterialized venous blood has been drawn on the morning of surgery.

DR. CLARENCE E. DENNIS, Pelham Manor, N. Y.—I should be honest and state that it is not only paper No. 25 that I wish to discuss, but the whole problem of the pump oxygenator. There are several items which, from the experience my associates and I have had first at Minnesota and now in Brooklyn, may throw light on the whole problem.

In the first place, it is not necessary to take all the blood which returns through the venae cavae, and the idea of taking only part of it, as Dr. Lillehei has emphasized, is very important and very helpful. As a consequence, we take blood from the venae cavae only by gravity. In the second place, we found we were having trouble with air embolization, and a review of the data of many others working on this has led us to believe that they also had air embolization that, perhaps, was not recognized. In the third place, the problem of preventing clotting in the apparatus, without having trouble with postoperative hemorrhage, has a very real bearing on the success of this approach to the problem. We find that by utilizing protamine titration repeatedly during the course of the operation, obtaining heparin concentrations of approximately 50 μg per cubic centimeter, by back titration with protamine as soon as perfusion is finished, and by fresh blood transfusions that amount to about one-third of the blood volume at the end of the procedure, we are able to return the clotting mechanism to a condition that is essentially normal.

In the fourth place, the addition of reduction of body temperature seems to have some beneficial effect on the procedure. We find that trying to keep the temperature below 90° F. has been, in dogs, associated with more complications than benefits, and that by carrying the temperature down to approximately this level and using for the purpose of perfusion about 65 c.c. of blood per kilogram, we have been very successful.

In the fifth place, the matter of the flowmeter; Dr. Senning in Dr. Crafoord's laboratory designed a flowmeter which has been copied by Dr. Newman in our laboratory. This works very beautifully indeed, and seems to be accurate within 2 or 3 per cent.

Finally, it seems to us that the apparatus does not need to be complicated. (slide) This is the apparatus that we used on 1 patient in whom we were able to clear pulmonary edema and hydrothorax very satisfactorily when it had not been possible to do so by other means. When the blood comes out of the femoral vein in this instance it goes through the flowmeter and oxygenator by gravity, and then is pumped through a bubble remover back to the patient. Finally, there has been developed a rather critical design of bubble remover which is very important in our opinion. It has been effective in taking all bubbles from the blood. There are no vacuum tubes in the circuit, the pump fills by gravity, and no part of the system has negative pressure between that point and the patient.

Of the dogs that have been perfused in the laboratory and have had ventricular septal defects made, as Dr. Newman showed in his motion picture yesterday, in the last 25 animals there have been 23 long-term survivals. The 2 deaths were unrelated to the perfusion process. The last 10 dogs have all survived and appear to be in good health. When we use dogs under 6 kilograms in weight, however, new problems arise which we still have to solve.

THE ARTERIALIZATION OF BLOOD AS IT APPLIES TO THE MECHANICAL HEART-LUNG APPARATUS

F. D. Dodrill, M.D., Alfred Lui, M.D., (by invitation), Jan Nyboer, M.D. (by invitation), E. V. Rippingille (by invitation), and C. H. Hughes, B.S. (Med. Biol.) (by invitation)
Detroit, Mich.

THERE is a very great need for a method of arterialization of the blood as it is passed through a pumping device so that the total venous return from the heart can be diverted through such an apparatus and returned to the arterial system as good arterialized blood. There are two principal methods by which this can be accomplished. The blood can be spread out as a thin sheet of red cells which are exposed to oxygen and which also allows the escape of the carbon dioxide. This involves a large cross-sectional area and in order to fill such a device large quantities of blood are required. With the use of such an apparatus, the total blood volume in smaller animals and in patients is completely removed as donor blood replaces it. The other way by which the blood may be arterialized is by the gaseous dispersion of oxygen through the blood. This brings a huge number of small oxygen bubbles through the blood so that instead of spreading out the blood in a large film, the oxygen is spread out over a large surface area. The former method has been extensively investigated by Gibbon and his associates.[1] The latter method has been extensively investigated by Clark, Gollan, Gupta,[2] and others. The latter investigators used porcelain plates and bacteriologic filters. It is the purpose of this manuscript to report a different type of dispersion arterialization.

THE ARTERIALIZER

Oxygen is passed upward through a stainless steel plate which is perforated with numerous tiny holes of 10 microns in diameter, Fig. 1. The completely assembled apparatus is diagrammatically illustrated in Fig. 2. Oxygen, which enters at *A*, is provided at a given pressure below the plate, *C*, which is placed transversely. The tiny oxygen bubbles emerge from the top of the plate and are brought in contact with the blood. Blood enters the bottom of the apparatus, *B*, where it produces a swirling motion throughout the annular surface of the chamber. During this swirling motion, the blood and the tiny bubbles are brought in close approximation. The metallic cylinder, *D*, is simply a variable space-occupying piece of metal which forces the blood and oxygen to pass up the annular surface, *E*. As the blood and oxygen mixture reaches the level *F*, annular perforations are present at this point whereby the blood enters the coalescent chamber, *G*. Here it is in contact with organo-polysiloxane which

From the Research Division of Harper Hospital and the Research Laboratories Division, General Motors Corporation.

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is commonly known as Dow-Corning Antifoam A. This substance changes the surface tension of the minute bubbles causing their coalescence and the large bubbles of excess oxygen and carbon dioxide escape at the outlet tube, *J*, as the blood spills over in the collecting chamber, *H*, where it is removed through a dependent tube, *I*. The rate of arterialization can be regulated by the pressure beneath the metallic plate. The amount of excess oxygen used is not great. This may be only about 40 to 50 per cent in excess of the actual requirements of the body.

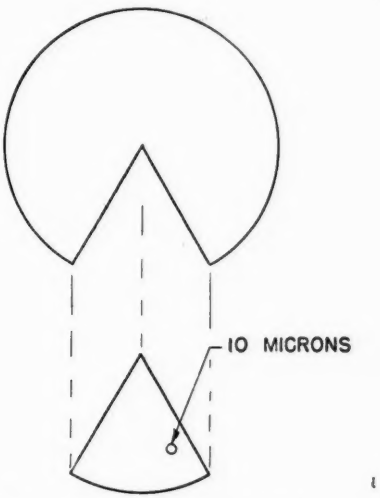


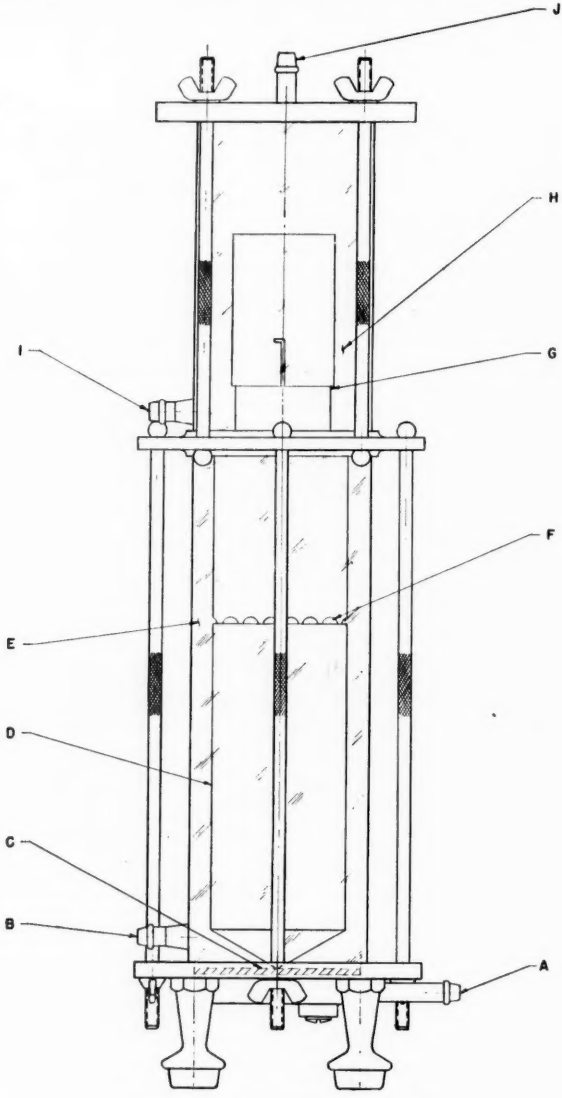
Fig. 1.—Porous stainless steel plate.

Metallic plates of 10 microns in diameter give a porosity of 45 per cent. Plates with different sized holes may be obtained. Although smaller holes and, therefore, smaller bubbles of oxygen can be obtained, the amount of carbon dioxide released and the alternation in blood pH seems to be directly in relation to the size of the holes.

We have found during numerous experiments that if 100 per cent oxygen is bubbled through the plate in sufficient quantities to fully arterialize the venous blood, the amount of carbon dioxide which will be washed away by the process is in excess of normal. The result is that excess carbon dioxide is washed out, the carbon dioxide content of the arterialized blood is lower than normal, and the pH is likewise altered. For this reason, instead of using 100 per cent oxygen as the dispersion gas, we are currently using a mixture of 98 per cent oxygen and 2 per cent carbon dioxide. With the use of this mixture, excess carbon dioxide is not removed from the blood while adequate arterialization takes place. The pH, therefore, is not markedly altered in the process.

Connection to the Mechanical Heart.—Our mechanical heart has been previously described[3] and consists of two sides which simulate the two ventricles. The arterializing apparatus is simply attached between the two ventricles as

the lungs are situated between the right and left heart. The right side of the pump removes the blood from the venae cavae, pumps it through the arterializer and the left side of the mechanical heart picks up the arterialized blood and returns it to the arterial system. Figs. 3*A* and 3*B* illustrate the total apparatus consisting of the two sides of the pump together with the arterializer.



APPARATUS FOR THE ARTERIALIZATION OF BLOOD

Fig. 2.

RESULTS OF EXPERIMENTS

The apparatus is connected to the animal by cannulating both venae cavae for the inflow and the left subclavian artery and left femoral artery for the outflow. The venae cavae are ligated around the tubes and the azygos vein is occluded. All return to the right heart must, therefore, pass through the apparatus. Extensive determinations of the arterial oxygen saturation, carbon dioxide content, and pH of the arterialized blood have been made. In addition, blood counts, platelet counts, and fragility tests have been made.

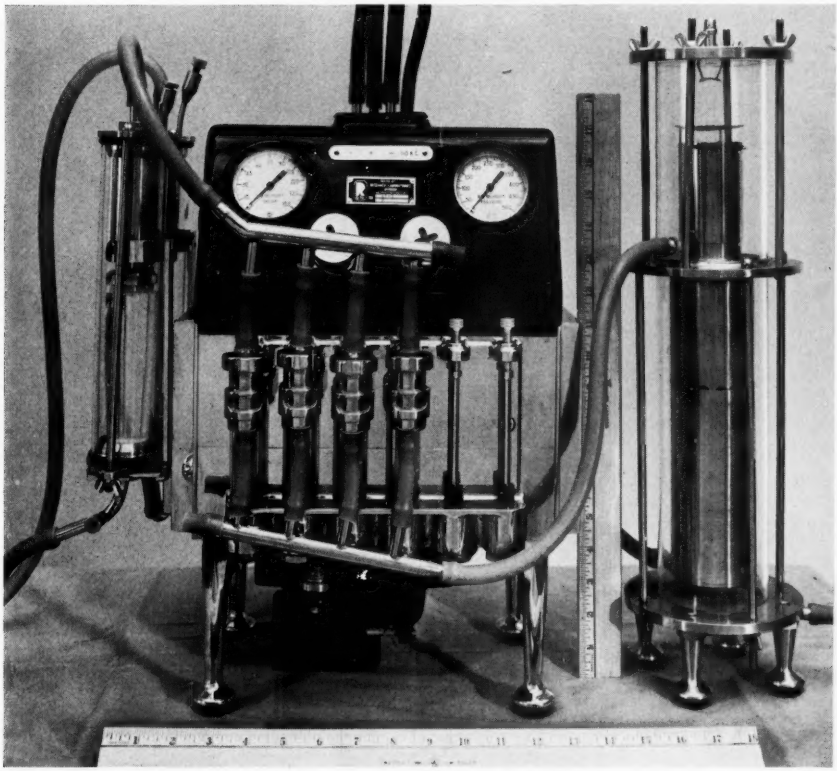


Fig. 3A.—Photograph of the apparatus consisting of the pump together with the arterializer; front view.

Oxygen Saturation.—Fig. 4 illustrates a scatter graph in which the oxygen saturation of the blood after it has passed through the apparatus is recorded at approximately 5 minutes, 10 or 15 minutes, and at 30 minutes of total bypass. It can be seen at a glance that the oxygen saturation is quite adequate in practically each and all instances. The problem, therefore, of adding sufficient oxygen to the venous blood seems not to be a serious one. As the pumping continues, the temperature of the blood is decreased by the extracorporeal

circulation so that arterialization in excess of 100 per cent saturation is common. We believe that minute bubbles of oxygen are not present in the blood as it escapes from the arterializer. In the event that free bubbles could be present, however, there is adequate provision for them to escape as the blood passes through the left side of the mechanical heart and through the flowmeter and bubble trap. It can be seen from the graph on the oxygen saturation that out of approximately 30 determinations in 10 experiments, practically all are of satisfactory saturations.

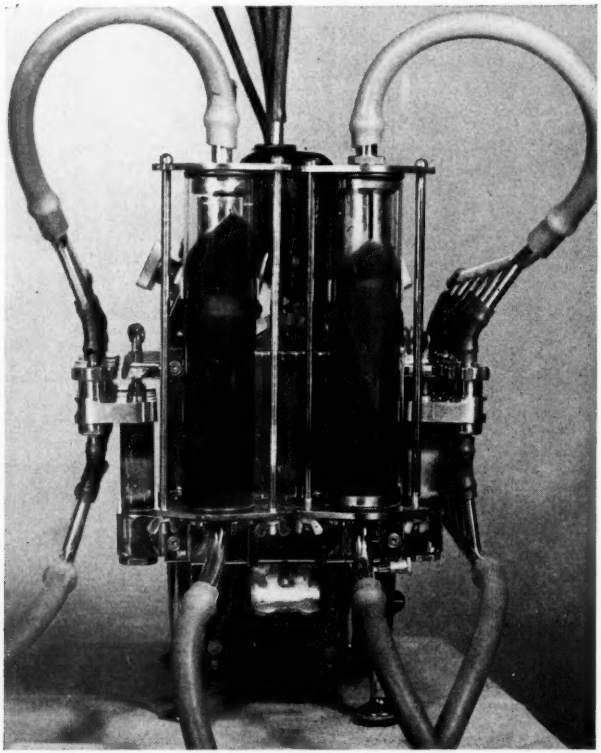


Fig. 3*B*.—Photograph of the apparatus consisting of the pump together with the arterializer; side view.

Carbon Dioxide Content.—With the use of 98 per cent oxygen and 2 per carbon dioxide, excess carbon dioxide is not removed as it passes through the arterializer so that the arterialized blood contains essentially normal carbon dioxide. Fig. 5 illustrates the arterial carbon dioxide in volumes percentages during 30 minutes of total by-pass. It can readily be seen that of approximately 30 determinations only a few can be regarded as abnormal.

pH Determinations.—Fig. 6 illustrates a scatter graph in which 10 experiments are carried out during which time approximately 30 determinations of

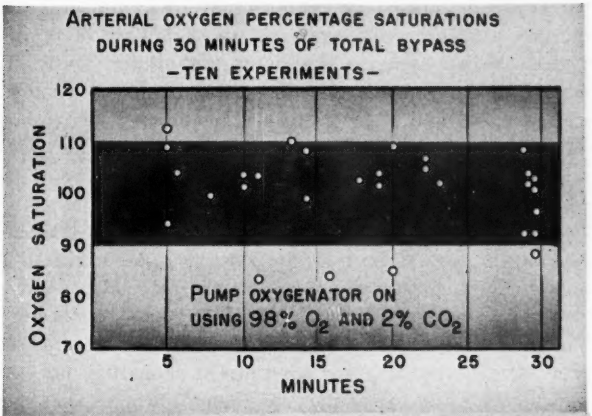


Fig. 4.

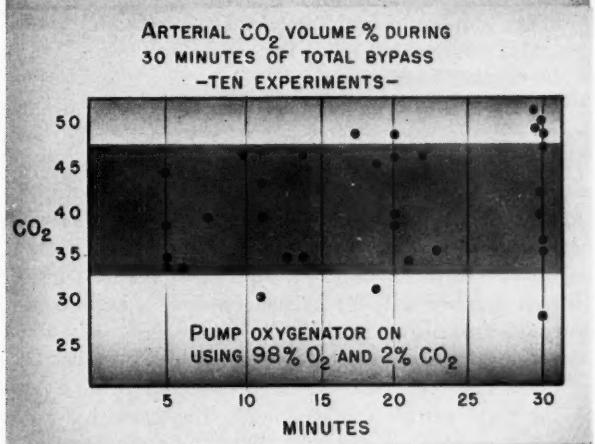


Fig. 5.

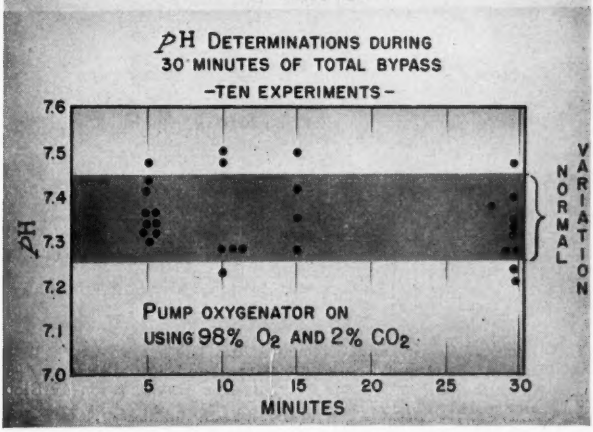


Fig. 6.

Figs. 4-6.—Described in text.

the pH are made at approximately 5 minutes, 10 to 15 minutes, and at 30 minutes' duration. A glance at the graph shows that for a 30-minute period of complete by-pass the pH remains within normal limits. It is interesting to note that the pH of the blood of the dog, after it has been anesthetized, the chest has been opened, and it is on forced respiration, shows a tremendous variation. We usually find at this time that the pH may be greatly elevated to 7.6 or 7.8 but that immediately after it passes through the apparatus it has been reduced to a normal level and remains essentially so throughout the 30 minutes of complete by-pass. Many factors are involved in the pH of the blood aside from the actual process of passing it through the arterializer. Such things as hemorrhage, transfusion, and the arterial pressure are factors causing variations. Prolonged pumping of blood leads eventually to the destruction of blood elements. The first constituent to be affected are the platelets. Following 30 minutes of pumping, there is some diminution in platelets, which may be as high as 50 per cent. We have also found that the eosinophils disappear early in the pumping period. Fragility tests and total blood counts have not been appreciably changed. Free hemoglobin for a 30-minute by-pass is usually not in excess of 100 to 150 mg. per cent.

Given arterialized blood of normal oxygen content, of normal carbon dioxide content, and the acidity adjusted to a normal pH, the perfusion of such blood into the arterial system in such a way as to provide an adequate arterial pressure provides an excellent chance for the opening of the heart and the performance of intracardiac procedures. We believe that not enough stress has been placed upon the arterial pressure. We like to maintain an arterial pressure of 110 to 120 mm. of Hg and, if one can do so using good arterialized blood, it seems unlikely that much serious harm could come to the body, especially during a short period of total by-pass. Our experiments are only for a 30-minute by-pass. We believe this time is sufficient for most intracardiac procedures under vision. We have been able to do a total by-pass in the dog with the right ventricle open for 30 minutes with a recovery rate of 75 per cent.

SUMMARY

A means of arterializing the blood doing a total by-pass is presented. The basic principle is the passing of oxygen and carbon dioxide through a metallic plate with holes of 10 microns in diameter. This produces tiny bubbles which come in contact with the venous blood by a swirling action. Instead of using 100 per cent oxygen, which removes excess carbon dioxide, we use 98 per cent oxygen and 2 per cent carbon dioxide. The addition of 2 per cent carbon dioxide to the oxygen keeps the carbon dioxide at essentially a normal level, and, therefore, the pH is not excessively altered.

REFERENCES

1. Miller, B. J., Gibbon, J. H., Jr., and Gibbon, M. H.: Recent Advances in the Development of a Mechanical Heart and Lung Apparatus, Ann. Surg. **134**: 694, 1951.
2. Clark, L. C., Jr., Gollan, F., and Gupta, V. B.: The Oxygenation of Blood by Gas Dispersion, Science **111**: 85, 1950.
3. Dodrill, F. D., Hill, E., and Gerisch, R.: Some Physiologic Aspects of the Artificial Heart Problem, J. THORACIC SURG. **24**: 134, 1952.

PULMONARY VENTILATION DURING OPEN THORACOTOMY

Inflation and Deflation Time Ratios and Pressures

Thomas F. Nealon, Jr., M.D. (by invitation), George J. Haupt, M.D. (by invitation), Joyce E. Price, R. N. (by invitation), and John H. Gibbon, Jr., M.D.
Philadelphia, Pa.

RESPIRATORY acidosis is a common occurrence in operative procedures.[4, 5, 8] The difficulty in achieving adequate pulmonary ventilation is increased when the thoracic cavity is opened.[2, 6, 10] Manual or mechanical compression of the rebreathing bag, providing intermittent positive pressure, has been used to improve ventilation during operation. A previous report from this laboratory[1] indicated that intermittent positive and negative pressure ventilation was more effective in preventing respiratory acidosis than either of the above methods.

The present report is a further study of intermittent positive and negative pressure ventilation. It is chiefly concerned with the effect upon pulmonary ventilation of the relative duration of inflation and deflation and of the pressures employed. Twenty patients were studied during twenty-one operations. All operations involved an open thoracotomy. The following measurements were made: the pH and carbon dioxide content of arterial blood, the concentration of carbon dioxide in the inspired and expired gas, and the pulmonary ventilation.

METHODS

A positive and negative pressure ventilator* (Fig. 1) was attached to the anesthesia machine. Carbon dioxide concentrations of the inspired and expired gases were measured by a Liston-Becker infrared absorption carbon dioxide analyzer.† The infrared analyzer was connected directly to the endotracheal tube. The apparatus was calibrated prior to use with gas samples of known carbon dioxide concentration which had been checked by the micro-Scholander technique.[9] Concentrations were recorded in per cent and converted to mm. Hg by using the formula: (barometric pressure—47 mm. Hg) $\times$ $\%CO_2 = pCO_2$. The calibration remained constant over long periods of time provided the instrument had been warmed up for several hours prior to use. Endotracheal pressures were read from an aneroid manometer. A dry test volume displacement

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*Jefferson Ventilator supplied through the courtesy of Air Shields Corporation, Hatboro, Pa.

†Liston-Becker Instrument Co., Inc., 649 Hope Street, Springdale, Conn.

gas flow meter* was placed in the expiratory tubing. This meter has a flow rate capacity of 2.4 to 70 L. per minute with a resistance of less than 0.5 cm. of water at the higher flow rate.

Arterial blood samples were obtained through a Cournand needle in the radial or brachial artery. Specimens were collected in oiled syringes containing a small quantity of heparin and a drop of mercury to aid in mixing the blood.

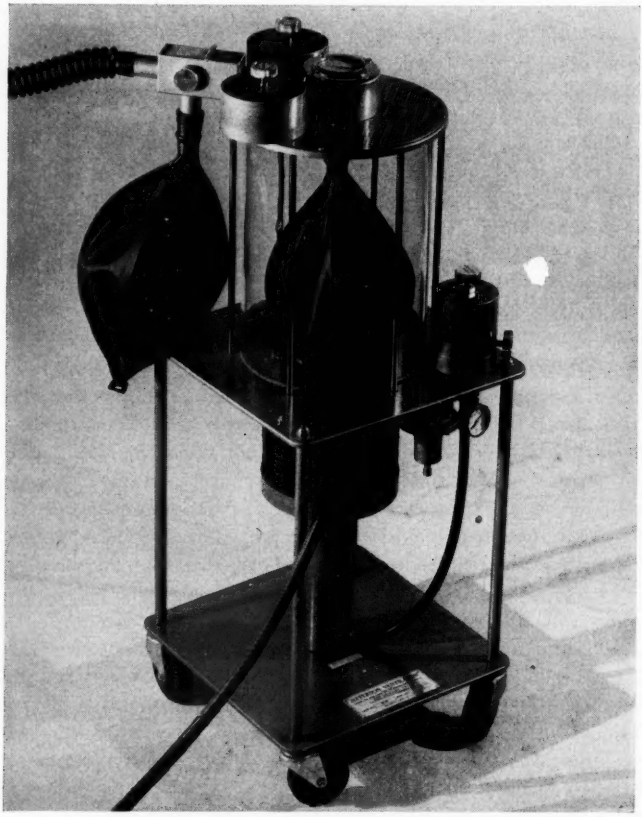

Fig. 1.—Photograph of the ventilator employed. The respiratory tubing is attached to the anesthesia machine in place of the rebreathing bag, which is reattached to the ventilator outside the glass cylinder (left of photograph). A switch at the top of the ventilator permits instantaneous change from unassisted, or manually assisted, ventilation with the bag outside the cylinder to automatic intermittent positive and negative ventilation with the bag inside the cylinder. The latter bag is alternately compressed and expanded by the action of the bellows beneath the cylinder.

Determinations of pH were made promptly after the withdrawal of the blood, using a Cambridge glass electrode pH meter calibrated with standard buffer solution before each determination. Duplicate determinations were made on each specimen and checks to 0.02 pH unit were required. The carbon dioxide

*American Meter Co., Albany, N. Y.

content of whole blood was determined using the technique of Van Slyke and Neill[11] modified to compensate for the presence of small amounts of ether as described by Goldstein and co-workers.[7] The plasma carbon dioxide content was derived from the line chart of Van Slyke and Sendroy.[12] The partial pressure of the carbon dioxide was then read from the nomogram of Van Slyke and Sendroy using the determined total plasma carbon dioxide content and pH values. In some cases arterial blood samples were obtained the day before operation and again before anesthesia was begun. A sample was drawn immediately after the endotracheal tube was inserted, and at intervals throughout the operation.

Preoperative sedation consisted of a barbiturate two hours before and morphine one hour before operation. Anesthesia was induced with nitrous oxide and oxygen and maintained with ether and oxygen. Case 18 was anesthetized with thiopental sodium alone. A size 7 mm. or 9 mm. endotracheal tube was inserted. Eighteen operations were performed with the patients in the lateral position and three with the patients in the supine position (Cases 3, 8, and 18).

Respiration was manually assisted until the trachea was intubated. Thereafter, positive and negative pressure ventilation was employed. No curare-like drugs were administered. Spontaneous respiratory activity ceased promptly after positive and negative pressure ventilation was begun, except in two instances (Cases 4 [a], and 13). When positive and negative pressure ventilation was discontinued, the patients were usually apneic for about one minute.

A ventilatory rate of 20 per minute was used in all patients. Three time ratios of inspiration to expiration were used: 1:2, 2:1, and 1:1. The effect upon ventilation of different inflation and deflation pressures was studied during the use of each of these three time ratios. The surgeon was not advised of the changes, but was requested to report the degree of expansion of the lung throughout the operation.

RESULTS

The effect upon pulmonary ventilation of varying the time ratio of inflation to deflation in 4 cases is shown in Table I. In this, and subsequent tables and

TABLE I

CASE NUMBER	RELATIVE DURATION INFLATION: DEFLATION	PRESSURE		PRESSURE DIFFERENCE cm. H_2O	MEAN PRESSURE cm. H_2O	VENTILATION L./MIN.
		INFLATION cm. H_2O	DEFLATION cm. H_2O			
4 (b)*	2:1	+20	−5	25	11.6	19.0
	1:1	+20	−5	25	7.5	22.0
18	1:2	+14	−7	21	0.0	14.0
	1:1	+15	−5	20	5.0	16.0
19	1:2	+15	−5.5	20	1.6	15.3
	2:1	+10	−10	20	3.3	17.1
	2:1	+15	−5	20	8.3	17.3
	1:1	+15	−5	20	5.0	18.9
20	1:2	+15	−5	20	1.6	15.7
	2:1	+15	−5	20	8.3	18.8
	1:1	+15	−5	20	5.0	21.5

*Case 4 was operated on two times, (a) and (b).

graphs, the recorded pulmonary ventilation is that which was actually measured. The figures have not been corrected for temperature and pressure, nor have they been related to the body surface area. The figures for pulmonary ventilation, therefore, may be compared with one another in the same patient, but are not comparable with the measured ventilation of other patients with different surface areas. With a constant inflation–deflation pressure difference and avoidance of collapse or overexpansion of the lung, the best ventilation was obtained when inflation and deflation were of equal duration (Table I). However, adequate pulmonary ventilation could be provided with an inflation to deflation time ratio of 2:1 (Cases 4 [b], 19, and 20), but it was difficult to provide adequate ventilation during an open thoracotomy with a time ratio of 1:2 (Cases 18, 19 and 20).

Table II lists measurements of pulmonary ventilation with different pressure spans during a constant time ratio of inflation to deflation. Nine patients were studied. In each case the pulmonary ventilation varied directly with the difference in pressure between inflation and deflation provided the lung was neither overexpanded nor collapsed.

TABLE II

CASE NUMBER	RELATIVE DURATION INFLATION: DEFLATION	PRESSURE		PRESSURE DIFFERENCE cm. H_2O	VENTILATION L./MIN.
		INFLATION cm. H_2O	DEFLATION cm. H_2O		
3	2:1	+8	−8	16	11.9
		+14	−8	22	15.0
		+16	−10	26	16.8
4 (a)	2:1	+10	−5	15	10.6
		+10	−8	18	12.9
		+10	−10	20	13.7
		+15	−10	25	15.8
5	2:1	+10	−10	20	11.7
		+15	−10	25	16.2
6	2:1	+15	−6	21	12.6
		+15	−8	23	13.9
		+15	−10	25	14.3
		+17	−8	25	14.4
9	1:1	+17	−7	24	20.0
		+19	−5	24	21.0
		+19	−7	26	24.0
11	1:1	+18	−5	23	16.5
		+18	−10	28	19.0
13	1:1	+15	−10	25	18.5
		+15	−12	27	19.5
		+17	−15	32	21.5
19	1:1	+10	+1	9	11.3
		+15	+2	13	15.6
		+15	−5	20	18.6
20	1:1	+15	+4	11	15.6
		+15	−5	20	21.5

Figs. 2, 3, and 4 are graphic illustrations of the studies made on 1 patient (Case 19) (Tables I and II). This man had pulmonary tuberculosis confined to the right upper lobe. The remainder of the lung was normal. Diagrammatic representations of the pressures and the timing of one respiratory cycle are shown in the upper half of each figure. The horizontal dotted line indicates the mean pressure during the respiratory cycle. The condition of the lung as

noted by the surgeon is indicated by the capital letters adjacent to the horizontal dotted line. When the mean pressure was in the neighborhood of 5 cm. of H_2O, the lung appeared to the surgeon to be properly expanded and yet it was soft and could be retracted easily. This is indicated by the letter *S*. The letter *T* indicate a tense, overexpanded lung and the surgeon complained of the difficulty in obtaining good exposure of the operative field. Every time such a complaint was made, the mean pressure was 7 cm. or above. When the mean pressure fell below 3 cm., the surgeon observed that although the lung expanded partially

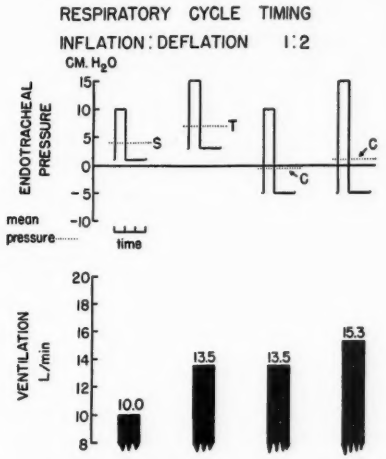

Fig. 2.—Graph of the ventilation in Case 19 with four different combinations of inflation and deflation pressures using an inflation to deflation time ratio of 1:2. The pressures used are illustrated diagrammatically for one respiratory cycle. The mean pressure is indicated by a dotted line. *S* indicates a soft, easily retractable lung which is normally expanded. *T* indicates a tense or overdistended lung. *C* indicates a collapsed lung.

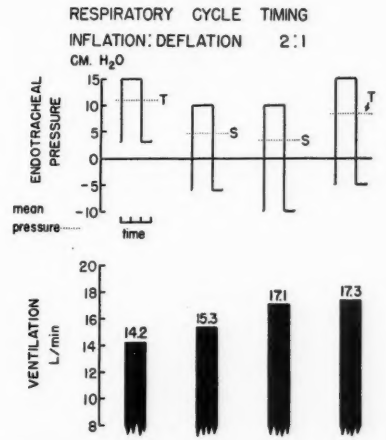

Fig. 3.—Graph of the ventilation in Case 19 similar to Fig. 2, but with an inflation to deflation time ratio of 2:1.

TABLE III

CASE NUMBER	DIAGNOSIS	OPERATION	DURATION OF OPERATION	THE pH AND pCO_2 OF ARTERIAL BLOOD					
				AFTER ENDOTRACHEAL INTUBATION		AVERAGE DURING OPERATION WITH VENTILATOR		END OPERATION SPONTANEOUS RESPIRATION	
				pH	pCO_2	pH	pCO_2	pH	pCO_2
1	Bronchogenic carcinoma	Lobectomy with partial excision of chest wall	4°33′			7.42	28.8	7.30	42.0
2	Bronchogenic carcinoma	Exploratory thoracotomy	2°20′	7.25		7.33	44.0	7.36	40.0
3	Pulmonary tuberculosis	Lobectomy	6°33′	7.28	61.0	7.37	48.1	7.33	47.0
4 (a)	Bronchiectasis	Lobectomy and segmental resection	4°40′	7.20	77.0	7.22	64.8	7.23	61.6
(b)	Bronchopleural fistula	Closure	4°37′			7.37	32.8	7.31	44.5
5	Bronchogenic carcinoma	Pneumonectomy	3°42′	7.26	63.0	7.36	42.8		
6	Pulmonary tuberculosis	Lobectomy	3°55′			7.38	33.0	7.34	41.0
7	Intrathoracic neurofibroma	Excision	2°36′			7.31	39.8	7.32	37.6
8	Myasthenia gravis	Thymectomy	2°30′	7.32	50.8	7.45	28.4	7.45	26.8
9	Neurofibroma of diaphragm	Excision	2°50′	7.32	52.0	7.42	34.5	7.41	38.5
10	Bronchiectasis	Lobectomy and segmental resection	3°55′	7.24	72.0	7.36	41.0	7.32	37.5
11	Bronchogenic carcinoma	Pneumonectomy	4°38′	7.29	41.6	7.34	34.7	7.35	36.8
12	Anthracosilicotic granuloma	Exploratory thoracotomy	3°28′	7.32	50.0	7.30	45.5	7.24	41.0
13	Pulmonary tuberculosis	Lobectomy	3°50′	7.17	82.0	7.29	59.3	7.38	51.0
14	Perinephric abscess with pleural effusion	Exploratory thoracotomy drainage of abscess	3°40′	7.40	40.0	7.49	30.6	7.44	35.0
15	Bronchogenic carcinoma	Exploratory thoracotomy	2°10′	7.27	52.0			7.34	39.5
16	Tuberculoma	Wedge resection	1°45′	7.34	35.5	7.32	37.6	7.29	41.3
17	Bronchogenic carcinoma	Exploratory thoracotomy	2°10′	7.29	55.0	7.33	43.1	7.40	41.0
18	Atrial septal defect	Closure	4°30′						
19	Pulmonary tuberculosis	Lobectomy	3°11′						
20	Bronchiectasis	Lobectomy and lingulectomy	3°40′						

during inflation, it appeared to be collapsed during the remainder of the respiratory cycle. The bar graphs at the bottom of the figures indicate the measured pulmonary ventilation in liters per minute using the inflation to deflation time ratios indicated at the top of the figures and the pressures shown diagrammatically above each bar.

As might be expected with an open thoracotomy, it was difficult to produce as much pulmonary ventilation when the deflation period was longer than the period of inflation (Fig. 2). With an inflation pressure not higher than 15 cm. H_2O, the best ventilation was 15.3 L. per minute. When the inflation period was longer than the deflation period, the pulmonary ventilation was better (Fig. 3). Without producing a tense, overexpanded lung, the best ventilation was 17.1 L. per minute. The greatest pulmonary ventilation, 18.9 L. per minute, was achieved when inflation and deflation were of equal length (Fig. 4). This ventilation of approximately 19 L. per minute was achieved with an inflating pressure not greater than 15 cm. H_2O and with a normally expanded, yet soft, and retractable lung.

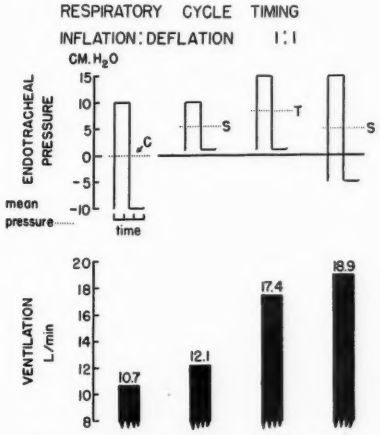

Fig. 4.—Graph of the ventilation in Case 19, similar to Figs. 2 and 3, but with an inflation to deflation time ratio of 1:1.

Table III lists the diagnosis, type, and duration of operation in all 20 patients. Case 4 was operated upon twice, (a) and (b). The pH and pCO_2 of arterial blood before positive and negative pressure ventilation was begun, the average of all determinations while the ventilator was in use and the values at the conclusion of the operation are shown. No arterial blood samples were obtained in Cases 18, 19, and 20.

Fig. 5 is a graph of these pCO_2 values and some preoperative values. *C* represents the mean value of all determinations. The pCO_2 was in the upper range of normal in the 6 patients in whom this determination was made the day before operation. Slightly higher values occurred in the 8 patients in whom the pCO_2 was measured just before induction of anesthesia, probably as a result of preanesthetic medication. The pCO_2 rose in all but 3 patients during induction

and in 6 patients was markedly elevated at the time of intubation. With the use of the ventilator, the arterial pCO_2 fell rapidly to near normal levels with the exception of 2 cases (*A* and *B*, Fig. 5).

In Case 4 (a) (*A* on Fig. 5) studied early in our experience, the elevated pCO_2 was not recognized. During a subsequent procedure, Case 4 (b) (Table III), the pCO_2 level was easily maintained below 40 mm. Hg. Case 13 (*B* on Fig. 5) was a short, stocky individual with severe pulmonary emphysema. He was markedly acidotic when the endotracheal tube was inserted: pH 7.17, pCO_2 82.0 mm. Hg. Throughout the operation, the pulmonary ventilation was 18 to 21 L. per minute. The pCO_2 of the arterial blood gradually fell but did not reach normal values. The greatest ventilation was obtained in this patient with a mean endotracheal pressure of +1. A high negative pressure was necessary to deflate the lungs (Table II).

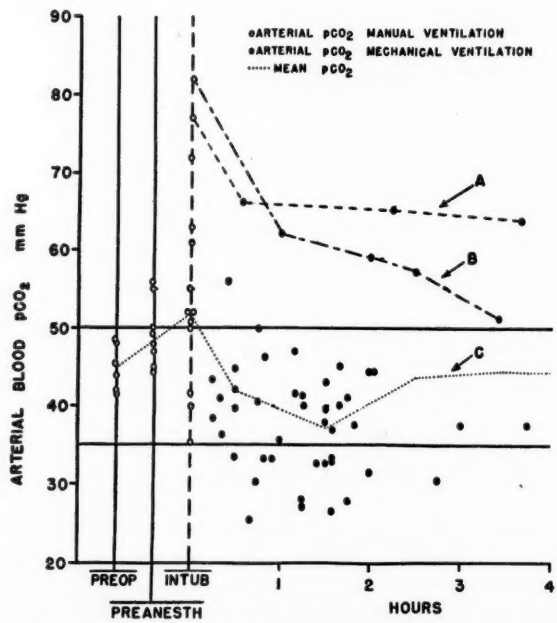

Fig. 5.—The pCO_2 of arterial blood during eighteen operations. In 6 patients this value was determined the day before the operation (*PRE-OP*). In 8 patients the pCO_2 was measured just before the induction of anesthesia (*PRE-ANESTH*). In 13 patients the pCO_2 was measured immediately following endotracheal intubation (*INTUB*). For explanation of *A* and *B*, Cases 4 (a) and 13, see text. *C* is the mean of all the points in the graph. The heavy horizontal lines indicate the upper and the lower limits of normal values.

The value of intermittent positive and negative pressure anesthesia is well illustrated in Case 3. A lobectomy for pulmonary tuberculosis was attempted twice, employing intermittent positive pressure anesthesia. On both occasions the operation was not begun because of a rapid pulse and a falling blood pressure following induction of anesthesia. When positive and negative pressure was used, satisfactory ventilation was achieved for over six hours (Fig. 6). In this

case, the pCO_2 of expired gas was measured by the Liston-Becker gas analyzer. The failure of the analyzer to record values of carbon dioxide equal to the levels in the arterial blood was probably due to technical factors which possibly can be eliminated.[3] Nevertheless the pCO_2 of the expired gas roughly paralleled changes in the pCO_2 of the arterial blood and provided a continuous indication that the pulmonary ventilation was adequate.

Another illustration of the value of continuous analysis of the expired gas is shown in Fig. 7 (Case 1). The per cent carbon dioxide in both the expired (top of tracing) and the inspired gas (bottom of tracing) is shown. Positive

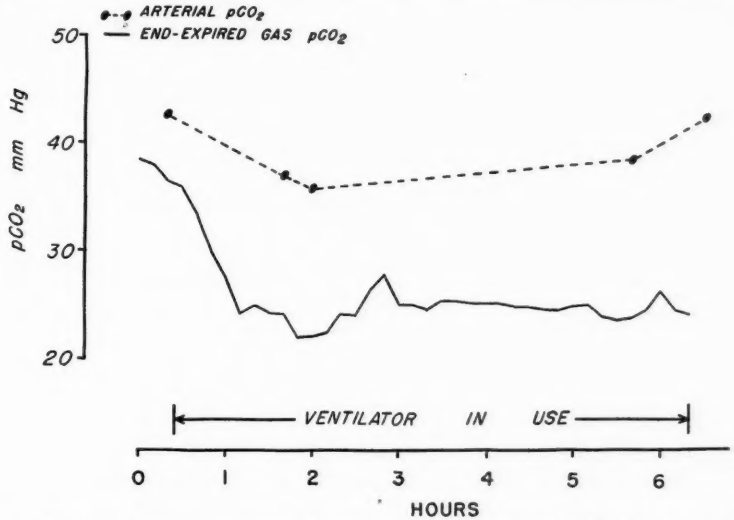

Fig. 6.—Graph of the pCO_2 of arterial blood and of the end expiratory gas during a six and one-half hour operation (Case 3).

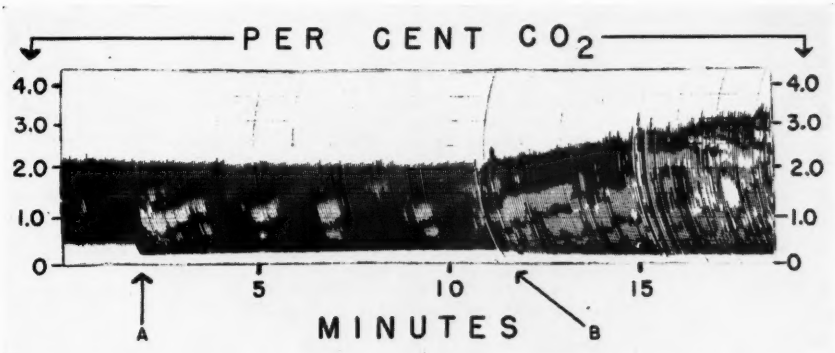

Fig. 7.—Photograph of a tracing made with an Esterline-Angus recorder showing the per cent carbon dioxide of the inspired and expired gas in Case 3. The top of the tracing indicates the per cent carbon dioxide of the expired gas; the bottom of the tracing indicates the per cent carbon dioxide of the inspired gas. At *A* the soda lime canister was changed. Positive and negative pressure ventilation was used until *B*. At *B* the ventilation was changed to manual intermittent positive pressure.

and negative pressure ventilation was used until point *B*. At point *A*, the soda lime canister was changed resulting in an immediate reduction in the per cent of inspired carbon dioxide. At *B*, the ventilation was changed to intermittent positive pressure at the same respiratory rate. The progressive rise in the per cent carbon dioxide of the expired gas is evident. A tracing such as this illustrates the value of continuous analysis of the expired gas in detecting poor function of the soda lime canister and the onset of respiratory acidosis. As in Fig. 6, the per cent carbon dioxide of expired gas, in this tracing, was below that of alveolar gas as shown by determination of the pCO_2 of arterial blood. Nevertheless the tracing reflected directional changes in the pCO_2 of arterial blood.

SUMMARY

1. With an open thoracotomy, the degree of expansion of the lung is a function of the mean endotracheal pressure within the obvious limits of collapse or overdistention. In the absence of generalized pulmonary disease, a mean endotracheal pressure of +4 to +6 cm. H_2O resulted in normal expansion of the lungs. With emphysema, lower mean pressures were required to avoid overexpansion of the lungs.

2. Avoiding collapse or distention of the lungs, with a respiratory rate of 20 per minute, and with a constant difference in pressure between inflation and deflation, the greatest pulmonary ventilation was achieved when the duration of inflation was equal to the duration of deflation.

3. Avoiding collapse or distention of the lung, with a respiratory rate of 20 per minute, and with equal duration of inflation and deflation, the pulmonary ventilation varied directly with the difference in pressure between inflation and deflation.

4. Greater pulmonary ventilation at a lower mean pressure can be achieved with the use of negative pressure during deflation. Negative pressure is also of great value in assisting the deflation of emphysematous lungs.

5. The use of positive and negative pressure ventilation resulted in normal or reduced levels of pCO_2 of arterial blood in 16 of 18 cases of open thoracotomy.

REFERENCES

1. Allbritten, F. F., Jr., Haupt, G. J., and Amadeo, J. H.: The Changes in Pulmonary Alveolar Ventilation Achieved by Aiding the Deflation Phase of Respiration During Anesthesia for Surgical Operations, Ann. Surg. **140**: 569, 1954.
2. Beecher, H. K., and Murphy, A. J.: Acidosis During Thoracic Surgery, J. THORACIC SURG. **19**: 50, 1950.
3. Collier, C. R., Affeldt, J. E., and Farr, A. F.: Continuous Rapid Infra-Red CO_2 Analysis. Fractional Sampling and Accuracy in Determining Alveolar Carbon Dioxide, J. Lab. & Clin. Med. **45**: 526, 1955.
4. Ellison, R. S., Ellison, L. T., and Hamilton, W. F.: Analysis of Respiratory Acidosis During Anesthesia, Ann. Surg. **141**: 375, 1955.
5. Gabbard, J. G., Roos, A., Eastwood, D. E., and Burford, T. H.: The Effect of Ether Anesthesia Upon Alveolar Ventilation and Acid Base Balance in Man, Ann. Surg. **136**: 680, 1952.
6. Gibbon, J. H., Jr., Allbritten, F. F., Jr., Stayman, J. W., Jr., and Judd, J. M.: A Clinical Study of Respiratory Exchange During Prolonged Operations With an Open Thorac, Ann. Surg. **132**: 611, 1950.
7. Goldstein, F., Gibbon, J. H., Jr., Allbritten, F. F., Jr., and Stayman, J. W., Jr.: The Combined Manometric Determination of Oxygen and Carbon Dioxide in Blood in the Presence of Low Concentrations of Ethyl Ether, J. Biol. Chem. **182**: 815, 1950.

8. Martin, F. E., MacDonald, F., and Stead, W. W.: Bronchospirometric Studies During Thoracic Surgery, J. THORACIC SURG. **29**: 327, 1955.
9. Scholander, P. F.: Analyzer for Accurate Estimation of Respiratory Gases in One-Half Cubic Centimeter Samples, J. Biol. Chem. **167**: 235, 1947.
10. Stead, W. W., Martin, F. E., and Jensen, N. K.: Physiologic Studies Following Surgery. IV. The Mechanism of the Development of Acidosis During Anesthesia, J. THORACIC SURG. **25**: 435, 1953.
11. Van Slyke, D. D., and Neill, J. M.: The Determination of Gases in Blood and Other Solutions by Vacuum Extraction and Manometric Measurement, J. Biol. Chem. **61**: 523, 1944.
12. Van Slyke, D. D., and Sendroy, J., Jr.: Studies of Gas and Electrolyte Equilibria in Blood. XV. Line Charts for Graphic Calculations by the Henderson-Hasselbalch Equation and for Calculating Plasma Carbon Dioxide Content From Whole Blood Content, J. Biol. Chem. **79**: 781, 1928.

DISCUSSION

DR. WILLIAM H. FALOR, Akron, Ohio.—We too have been interested in the problem of mechanical ventilation, and Drs. Ferro and Kelly on our service have studied an almost consecutive series of 136 patients, relative to carbon dioxide pile-up.

(slide) The first group was a series of 20 patients in whom the ordinary manual method of ventilation was employed. Carbon dioxide tensions were determined prior to anesthesia, at the time of pleurotomy, at the time of pleural closure, and at the end of the operation. In these 20 cases there was frequently a substantial carbon dioxide increase. Using the Rand-Wolfe breathing machine and a modified face-down position in 50 cases, we have commonly prevented respiratory acidosis. In 41 cases, using the lateral position with the machine, we have been equally effective in preventing acidosis.

(slide) The only complication attributable to mechanical ventilation that we have noted is the development in a few cases of superficial pressure necrosis of the pharyngeal mucosa. With each positive pressure phase of ventilation the endotracheal tube is forcibly introduced into the respiratory tract and in cases using an oral pack, pressure on the anterior pillars is transmitted by this pack. During a long operation, ventilation is carried out three or four or five thousand times and necrosis of the pharynx may develop. I would like to ask Dr. Gibbon if he has looked for or noted any evidence of tracheal necrosis developing as a result of the to-and-fro motion of the endotracheal balloon responding to the positive-negative phase machine.

DR. JOHN H. GIBBON, Jr.—The question was asked whether in using cuffed endotracheal tubes we had seen instances of necrosis of the tracheal mucosa. We have been using cuffs on endotracheal tubes now for many years, and we have seen no reason to abandon them. Dr. Crafoord says he has also used them in several thousand cases and has never seen any necrosis of the tracheal mucosa that he can remember. He mentioned, however, that necrosis might occur with prolonged use of such tubes for weeks or months in patients with poliomyelitis. I believe Dr. Beecher has seen an occasional case of necrosis, but we have not encountered it in our experience and we see no reason to abandon the use of the cuffed endotracheal tube.

Original Communications

LOBAR VENTILATION AND OXYGEN UPTAKE IN MAN

Influence of Body Position

Stig Börje Mattson and Eric Carlens
Uttran, Sweden

As early as the close of the nineteenth century it was suggested that the blood flow in different parts of the lungs varies to a certain extent with the position of the body. In conjunction the theory was advanced that anemia in the apices of the lungs when the posture is erect was the cause of "apical tuberculosis." This led to the introduction by different investigators of postural treatment in pulmonary tuberculosis. Bjure and Laurell[1] (1927) were of the opinion that they could demonstrate roentgenologically that the vascular flow in the uppermost portion of the lung was less in the standing than in the recumbent position. This condition they interpreted as an orthostatic arterial anemia. Bronchospirometry with the patient in the lateral recumbent position, which was first performed by Björkman[2] (1934) and subsequently repeated by numerous investigators, shows rather unequivocally that the lower lung increases its oxygen uptake at the expense of the upper, notwithstanding the fact that the ventilation often retains the same relation as in the supine position. Björkman suggests that "it is plausible that this is caused by increased perfusion in the lower lung and decreased in the upper resulting from the body position." Since cardiac catheterization has opened the possibility for measurement of the pressure in the pulmonary artery, interest has been renewed in the question of the significance of the orthostatic pressure to the pulmonary circulation. Taking as the point of departure, the normal pressure in the pulmonary artery and the estimated height of a column of fluid from the right ventricle to the apex of the lung, Dock[3] (1946) calculated that the apex in adults has little or no blood flow in the erect posture. Lagerlöf, Eliasch, Werkö, and Berglund[4] (1951) reached similar conclusions. Through comparative analysis of the alveolar air in different parts of the lung in man in the upright and recumbent positions Martin, Cline, and Marshall[5] (1953) arrived at results that may be interpreted as an effect of decreased pulmonary circulation in the right upper lobe in the upright posture. They showed that in this position the partial pressure of the oxygen was higher and that of the carbon dioxide was lower in the upper lobe than in the lower. In the recumbent position the relation between the gases in the two lobes was reversed. They write: "Whether this pattern is a result of ventilation and/or perfusion changes cannot be stated." Sadoul and Rahn[6] (1954) made similar observations on anesthetized dogs.

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The aim of the present study has been to determine spirometrically if and to what extent any shift occurs in the blood flow between the two regions of the lesser circulation above and below the pulmonary trunk when a man's position is changed from recumbent to erect. Anatomically the right lung is well adapted to such a dissociated spirometric investigation since, in the erect subject, the upper lobe is almost entirely above the level of the pulmonary trunk, and the two basal lobes are below. The construction of a suitable catheter has enabled us to measure the ventilation and oxygen uptake simultaneously in the upper and the two basal lobes of the right lung.

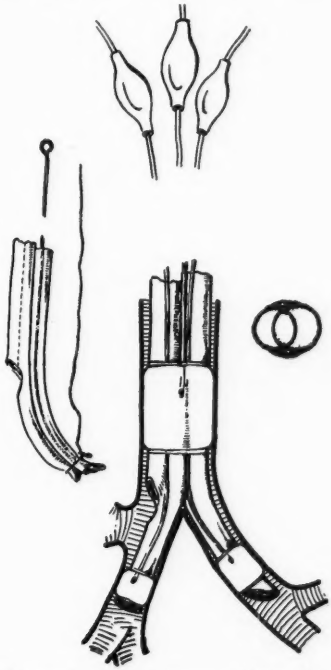

Fig. 1.—Diagram of the 3-lumen catheter in position.

MATERIAL

The investigation was carried out on 7 male patients between 21 and 37 years of age who were hospitalized for pulmonary tuberculosis. The right lung, which was the object of the investigation, exhibited no signs of pathologic change in any case. The pleurae and sinuses as well as the motility of the diaphragm on both sides were intact. In all cases there were parenchymal changes of moderate or slight extent within the upper part of the left lung. These were relatively fresh and not associated with coarse anatomic changes in the lung such as shrinkage or deformation of the central vessels. Case 2 is an exception. This patient had a pneumothorax about a fingerbreadth in extent on the left with exudate up to the cupola of the diaphragm. All cases reported here exhibited normal values in ordinary spirometry. Routine clini-

cal examination showed no indications of cardiac disease. On the basis of these observations, we feel justified in assuming that normal pressure existed in the lesser circulation. Patients with a wide laryngeal passage were selected, and in all cases the examination could be performed under correct technical conditions.

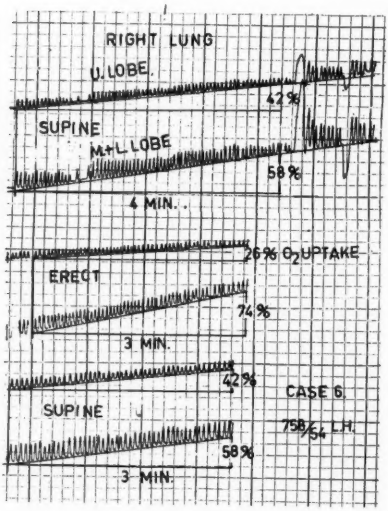

Fig. 2.—Spirometric tracings of different lobes of the right lung in supine and erect positions (Case 6).

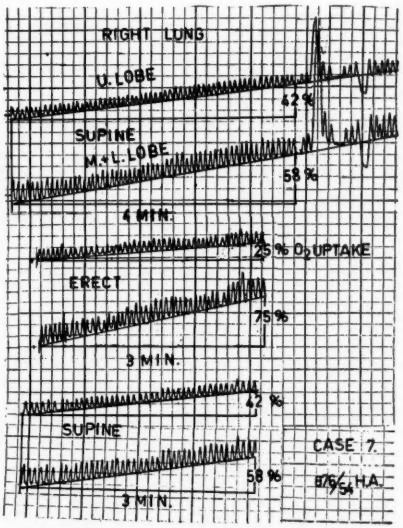

Fig. 3.—Spirometric tracings of different lobes of the right lung in supine and erect positions (Case 7).

METHODS

The patients received premedication consisting of 2 cg. Pantopon, 10 cg. luminal, and 1 mg. atropine administered subcutaneously 30 to 45 minutes prior to the topical anesthesia of the pharynx, larynx, and trachea with a small amount of 2 per cent tetracaine hydrochloride solution. The flexible catheter (Fig. 1) was passed under mirror guidance over a curved stylet through the larynx and down into the trachea. To facilitate the entrance the branches of the catheter were tied together with a slip knot. When the catheter was in the trachea, the stylet was removed and the knot was untied. The patient was placed supine on a table, and the correct position of the catheter was controlled fluoroscopically. After inflation of the cuffs, the channel to the right upper lobe was connected to one recording spirometer and the channel to the middle and lower lobes to another, both filled with oxygen. This twin spirometer is that originally described by Björkman (1934). The carbon dioxide in each circuit was absorbed by potassium hydroxide. In these experiments the ventilation of the left lung was not recorded. This lung was breathing ambient air, and the large tube in its main bronchus guaranteed free passage. The resistance to air flow in the catheter was investigated, and for the minute ventilation in question it never exceeded 0.7

cm. water. Before the recordings it was ascertained that the passage in the channels was free, by aspiration of any secretion that might be present. In none of the experiments did the patients exhibit any signs of dyspnea. After an observation period of several minutes during which the patient was allowed to reach a steady state, the oxygen uptake and ventilation were recorded during four minutes, first with the patient in the supine position. Thereafter the table was tipped to place the patient in the erect position, and similar recordings were taken during three minutes. As may be seen from Figs. 2 and 3, this period should be quite sufficient to give an accurate measure of the oxygen uptake. In several cases the patient was then tipped back into the supine position and a third set of recordings made to verify the accuracy of the first. The interval between recordings was about five minutes.

In order to ascertain that the catheter had remained in the correct position during the examination and that the passage to the respective lobe bronchi had been free, a bronchographic examination was carried out immediately after the recordings were finished. With a small quantity of contrast medium

Fig. 4.—A small quantity of contrast medium has been injected through the channel into the upper lobe after the spirometric examination to verify that free passage to this lobe had been maintained and that it was satisfactorily sealed off from other parts of the lung.

(Dionosil), it was first established that there was free passage to the upper lobe and that this lobe was satisfactorily sealed off from the other parts of the lung (Fig. 4). A corresponding examination of the middle and lower lobes was then performed (Fig. 5).

Determination of the vital capacity of the different parts of the lung, as seen in Figs. 2 and 3, may also be used to verify that free passage exists and that there is no leakage at the cuffs. If the breathing is stenotic, the

tracing does not instantly return to the respiratory mid-position after a maximum inspiration or expiration. There is a risk, however, that such a maximum respiratory movement may displace the position of the catheter.

Fig. 5.—After roentgen examination of the upper lobe (Fig. 4), the middle and lower lobes were similarly examined (lateral view).

RESULTS AND DISCUSSION

As is evident from Table I, the minute ventilation changed somewhat for the entire lung, but without any definite tendency, in the transition from supine to erect position. Under the same conditions the percentile share of the upper lobe in the ventilation showed an average decrease that was inappreciable ($m = 1.6$) but almost significant ($P<0.05$).

TABLE I. VENTILATION (LITERS PER MINUTE) OF RIGHT LUNG

CASE	AGE IN YEARS	ENTIRE LUNG		UPPER LOBE		MIDDLE LOBE + LOWER LOBE		UPPER LOBE EXPRESSED AS PERCENTAGE OF ENTIRE LUNG		
		SUPINE	ERECT	SUPINE	ERECT	SUPINE	ERECT	SUPINE	ERECT	DIFFERENCE
1. K. H.	25	7.7	8.4	2.6	2.8	5.1	5.6	34	33	-1
2. S. J.	35	10.0	9.1	4.4	3.6	5.6	5.5	44	40	-4
3. A. B.	28	5.9	7.4	2.5	2.9	3.5	4.5	42	40	-2
4. L. N.	37	9.1	9.0	3.6	3.6	5.5	5.4	40	40	0
5. B. K.	21	4.7	5.5	1.9	2.2	2.8	3.3	40	40	0
6. L. H.	23	3.8	3.4	1.4	1.2	2.4	2.2	37	35	-2
7. H. A.	24	4.6	4.9	1.8	1.8	2.8	3.0	39	37	-2

The total oxygen uptake in the right lung increased in the erect position in 6 cases, as may be seen in Table II. One of the patients (Case 3) was somewhat affected at the end of the examination. The values here deviated from the rest, showing a decrease in the total oxygen uptake in the erect position.

TABLE II. OXYGEN UPTAKE (LITERS PER MINUTE) OF RIGHT LUNG

	ENTIRE LUNG		UPPER LOBE		MIDDLE LOBE + LOWER LOBE		UPPER LOBE EXPRESSED AS PERCENTAGE OF ENTIRE LUNG			PERCENTILE DECREASE IN UPPER LOBE WITH CHANGE FROM SUPINE TO ERECT POSITION
CASE	SUPINE	ERECT	SUPINE	ERECT	SUPINE	ERECT	SUPINE	ERECT	DIFFERENCE	
1	0.236	0.250	0.073	0.030	0.163	0.220	31	12	-19	59
2	0.290	0.338	0.088	0.054	0.202	0.284	30	16	-14	39
3	0.221	0.159	0.086	0.039	0.135	0.120	39	25	-14	55
4	0.164	0.183	0.052	0.016	0.112	0.167	32	9	-23	69
5	0.225	0.263	0.095	0.040	0.160	0.223	42	15	-27	58
6	0.158	0.180	0.067	0.047	0.091	0.133	42	26	-16	30
7	0.188	0.200	0.078	0.050	0.110	0.150	42	25	-17	36

In all of the cases investigated, however, both an absolute and a relative decrease in the oxygen uptake in the upper lobe took place with a change from recumbent to erect position. The average percentile decrease was appreciable ($m = 18.6$) and highly significant ($P<0.001$). If we calculate the percentile decrease in the oxygen uptake in the upper lobe occurring with a change from supine to erect posture, we obtain values between 30 and 69. The basal lobes show a corresponding increase in the oxygen uptake under the same conditions.

Since the relative ventilation of the two parts of the lung undergoes only an inconsiderable change with a change in position, the great difference in the oxygen uptake must be an expression of a change in perfusion. Against the background of earlier observations presented in this article, it seems probable that it is the changed orthostatic pressure which causes the difference in the pulmonary circulation in the two positions. Inasmuch as the pressure in the pulmonary artery in the cases investigated here was assumed to lie within normal limits, the differences obtained in the oxygen uptake in the upper lobe would seem to be normal variations occurring with changes in position. In a pathologic rise or fall of the pressure, this difference may be expected to decrease or increase, respectively.

The greatly decreased perfusion with practically unaltered ventilation of the upper lobe occurring in the erect position should lead to an increase in the alveolar oxygen tension, which has already been demonstrated by Martin and associates,[5] and Sadoul and Rahn.[6] Therefore, in our opinion, the results of the present study appear strongly to support the old theory that the changed circulation in the apices of the lungs comprises a phthisiogenetic factor. That which is of essential significance is probably the increased oxygen tension in the tissues with more favorable growth conditions for the tubercle bacilli. Investigations by Scott, Hanlon, and Olson[7] support this theory. They found that anastomosis between systemic and pulmonary arteries in monkeys

infected intravenously with human tubercle bacilli results in an extensive caseous form of the disease in the lung on the side of the anastomosis.

The figures given here as a measure of the decreased oxygen uptake in the upper lobe must, of course, be regarded as mean values for the different parts of the lobe. It is therefore justifiable to assume that the venous blood flow in the apical parts is even less and perhaps, in certain cases, even totally absent. It is improbable, however, that the capillaries collapse and that the apex becomes completely empty of blood under these conditions, inasmuch as in experimental block of the venous circulation a flow of blood is very quickly obtained peripheral to the block with arterial blood from anastomoses with the bronchial arteries (Nordenström,[8] 1954).

As pointed out previously, when there is a pressure rise in the lesser circulation, the physiologic decrease of the blood flow in the apex in the erect position, which has been demonstrated here, may be expected to be moderated or entirely abolished. In the case of pathologically low pressure, this decrease should be more pronounced. Typical examples of such pathologic changes in pressure are found in mitral stenosis and in pulmonary stenosis, with increased pressure in the former and decreased in the latter. Moreover, it has long been known, and statistically verified by White[9] (1944), that pulmonary tuberculosis occurs less frequently in patients with mitral stenosis and considerably more frequently in those with pulmonary stenosis than in the population as a whole.

SUMMARY

The ventilation and oxygen uptake in the upper lobe and the basal lobes of the right lung was spirometrically investigated in 7 male patients. A comparative study showed an inconsiderable decrease in the ventilation (m = 1.6) but an appreciable decrease in the oxygen uptake (m = 18.6) in the upper lobe with a change from the supine to the erect position.

The decreased perfusion in the upper lobe is probably orthostatic in origin and may give rise to increased alveolar oxygen tension.

The significance of the changed circulation to the predominantly apical localization of postprimary tuberculosis is discussed.

REFERENCES

1. Bjure, A., and Laurell, H.: Om abnorma statiska circulationsfenomen och därmed sammanhängande sjukliga symptom. Den arteriella orthostatiska anemin en försummad sjukdomsbild, Uppsala Läkaref Förh. **33**: 1, 1927.
2. Björkman, S.: Bronchospirometrie. Eine klinische Methode, die Funktion der menschlichen Lungen getrennt und gleichzeitig zu untersuchen, Acta med. scandinav. suppl. **56**: 1934.
3. Dock, W.: Apical Localization of Phthisis, Am. Rev. Tuberc. **53**: 297, 1946.
4. Lagerlöf, H., Eliasch, H., Werkö, L., and Berglund, E.: Orthostatic Changes of the Pulmonary and Peripheral Circulation in Man, Scandinav. J. Clin. & Lab. Invest. **3**: 85, 1951.
5. Martin, C. J., Cline, F., and Marshall, H.: Lobar Alveolar Gas Concentrations: Effect of Body Position, J. Clin. Invest. **32**: 617, 1953.
6. Sadoul, P., and Rahn, H.: Relation ventilation-perfusion dans les differents lobes du chien anesthesié, Rev. med. Nancy **79**: 747, 1954.
7. Scott, H. W., Hanlon, C. R., and Olson, B. J.: The Effects of Experimental Alteration of the Pulmonary Arterial Circulation on Tuberculosis in Monkeys, Bull. Johns Hopkins Hosp. **88**: 376, 1951.
8. Nordenström, B.: Temporary Unilateral Occlusion of the Pulmonary Artery, Acta radiolog. suppl. **108**: 1954.
9. White, P. D.: Heart disease, ed. 3, New York, 1944, The Macmillan Company, p. 398.

THE USE OF "ARTERIALIZED" BLOOD FOR THE DETERMINATION OF ARTERIAL O_2 AND CO_2 TENSIONS

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INTRODUCTION

IN evaluation of pulmonary function, it may be helpful to know the arterial O_2 and CO_2 tensions. Björk[1, 2] has employed these determinations in the evaluation of pulmonary function by observing the change in O_2 and CO_2 tension levels after hyperventilation or exercise. The change in O_2 tension was studied rather than the change in O_2 saturation because in the physiological range a small alteration in O_2 saturation will be reflected in a larger and more easily detectable alteration in O_2 tension (Fig. 1).

In order to perform Björk's tests, it is necessary to do repeated arterial punctures, or to use an indwelling arterial needle to obtain the blood samples. We have sought a method of "arterializing" venous blood so that venepuncture might be substituted for arterial puncture. The latter is disagreeable to many patients and may be more hazardous.

Lilienthal and Riley[3] have shown that if an ear is heated by radiant heat for 10 minutes at an air temperature of 45° C. near the external ear, blood drawn from the ear lobe by puncture with a sharp-pointed knife will have the same O_2 saturation as brachial artery blood. Goldschmidt and Light[4] have shown that blood drawn anaerobically from a vein on the dorsum of a hand which has been heated to 45 to 47° C. for 10 minutes is equivalent to arterial blood in CO_2 and O_2 content.

Since the volume of blood obtainable by the method of Lilienthal and Riley is limited, and since repeated ear puncture is objectionable to many patients, we have chosen to use the method of Goldschmidt and Light to "arterialize" blood, and have determined the O_2 and CO_2 tensions of simultaneously drawn arterial and "arterialized" blood in a series of patients at rest. If the "arterialized" venous blood tensions approximated the arterial tensions under resting conditions, additional studies could subsequently be carried out to determine whether Björk's tests could be simplified by using "arterialized" rather than arterial blood.

METHODS

All subjects lay supine for 15 minutes before blood was drawn. The upper extremity was then immersed in water for 10 minutes to a point just above

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the elbow. The temperature of the water was maintained at 45 to 47° C. by adding hot water as needed. At the end of the 10-minute period, the hand was withdrawn from the water just enough so that venepuncture could be carried out on the dorsum. A 10 ml. sample of blood was withdrawn anaerobically by gentle aspiration, using a 20 ml. eccentric tip syringe with a No. 20 gauge needle. The dead space of the syringe and needle was filled with concentrated heparin (100 mg./ml.) prior to drawing the blood sample. After the blood was drawn, a small amount of mercury was aspirated into the syringe to aid in mixing, the syringe tip was plugged with a toothpick, and

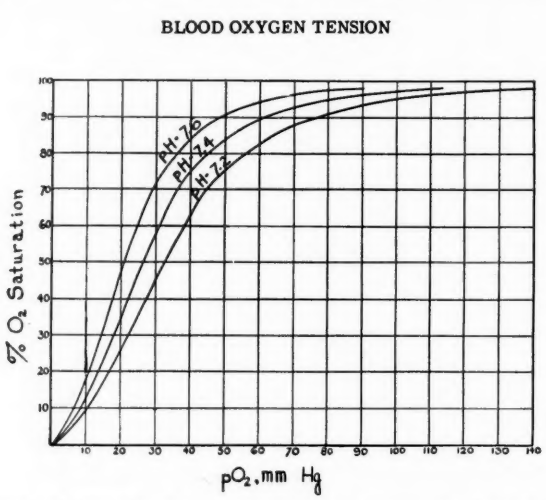

Fig. 1.—Oxyhemoglobin dissociation curve constructed from the data of Dill. (From J. H. Comroe, Jr., editor: Methods in Medical Research, Chicago, 1950, The Year Book Publishers, vol. 2, p. 163.)

the syringe agitated. Arterial blood was drawn at the same time from the contralateral brachial artery via a Cournand needle which had been placed in the artery. In three subjects a sample of venous blood was drawn within an hour from the same site as the "arterialized" blood, with the patient under similar resting conditions.

All blood samples were drawn without the use of a tourniquet. Analysis of blood samples was performed immediately. The CO_2 and O_2 tensions were determined with the Scholander blood gas tension apparatus by the method of Filley,[5] utilizing 30-unit Roughton-Scholander syringes. All tension determinations were carried out in duplicate, and checked within 6 mm. Hg. Tensions were expressed as the average of duplicate determinations.

Although we have not determined the correction factors for absolute values of the CO_2 and O_2 tensions by tonometry, the same correction factors apply to each pair of arterial and "arterialized" blood determinations and, therefore, the tensions in "arterialized" and arterial blood can be properly compared.

RESULTS

Determinations of O_2 and CO_2 tensions on brachial artery blood and "arterialized" venous blood drawn simultaneously from the dorsum of the hand were carried out under resting conditions in 14 male patients, some normal, and some with pulmonary disease. Similar studies were accomplished on 3 additional patients with blood drawn simultaneously from the brachial artery and a forearm or antecubital vein. None of the patients had any local disease which would have interfered with the peripheral vascular response.

Table I lists the CO_2 and O_2 tensions in the 17 patients.

TABLE I

CASE NO.	AGE	pCO_2 MM. Hg			pO_2 MM. Hg		
		"ARTERIALIZED"	ARTERIAL	DIFFERENCE	"ARTERIALIZED"	ARTERIAL	DIFFERENCE
1	59	32.0	31.8	+0.2	58.7	66.0	-7.3
2	63	50.7	49.8	+0.8	43.6	49.8	-6.2
3	61	38.8*	34.8	+4.0	51.2*	69.6	-18.2
4	58	43.1	38.0	+5.1	63.0	67.0	-4.0
5	61	38.8	36.2	+2.6	72.7	69.2	+3.5
6	65	40.2	42.1	-1.9	72.0	75.6	-3.6
7	58	40.5	44.3	-3.8	63.3	64.8	-1.5
8	29	42.9	42.6	+0.3	62.7	74.3	-11.6
9	44	43.4	47.8	-4.4	62.3	73.0	-10.7
10	26	40.1*	40.3	-0.2	80.8*	92.9	-12.1
11	64	43.4*	42.8	+0.6	66.0*	82.2	-16.2
12	24	45.5	43.1	+2.4	82.1	93.3	-11.2
13	33	51.6	40.0	+11.6	78.2	83.4	-5.2
14	63	44.9	46.8	-1.9	51.0	59.8	-8.8
15	53	45.2	40.5	+4.7	70.6	80.9	-10.3
16	41	45.2	46.6	-1.4	78.1	80.3	-2.2
17	28	34.4	34.6	-0.2	74.4	72.8	+1.6

*Blood drawn from forearm or antecubital region.

Table II presents a comparison of the "arterialized" and arterial blood gas tension findings in the group as a whole. The average determined CO_2 tension in "arterialized" blood was 1.1 mm. higher, and the average determined O_2 tension was 7.2 mm. lower than in arterial blood. The difference

TABLE II

	pO_2 MM. Hg	pCO_2 MM. Hg
Number of patients	17	17
Mean tension (avg.) in "arterialized" blood	66.6	42.4
Mean tension (avg.) in arterial blood	73.8	41.3
Difference of the means	-7.2	+1.1
Mean (avg.) of the differences (without regard to sign)	7.8	2.8
Extreme differences ("arterialized" minus arterial)	-18.2 to +3.5	-4.4 to +11.6

in O_2 tension of "aterialized" blood from that of arterial blood is much greater if "arterialized" blood is drawn from the forearm or antecubital region rather than from the dorsum of the hand, as shown in Table I, and as noted by Goldschmidt and Light. If only the "arterialized" samples drawn from the dorsum of the hand, and the corresponding arterial samples are compared, then the O_2 tension of the "arterialized" samples is on the average 5.5 mm. lower, and the CO_2 tension is on the average 1 mm. higher than that of arterial blood

TABLE III

	pO_2 MM. Hg	pCO_2 MM. Hg
Number of patients	14	14
Mean tension (avg.) in "arterialized" blood	66.6	42.7
Mean tension (avg.) in arterial blood	72.1	41.7
Difference of the means	-5.5	+1.0
Mean (avg.) of the differences (without regard to sign)	6.3	2.95
Extreme differences ("arterialized" minus arterial)	-11.6 to +3.5	-4.4 to +11.6

(Table III). The difference between "arterialized" and venous blood drawn from the same area at different times, but with the patient under the same resting conditions, gives an approximate indication of the extent of the change in venous blood produced by "arterialization" (Table IV).

TABLE IV

CASE NO.	pCO_2			pO_2		
	VENOUS	"ARTERIALIZED"	ARTERIAL	VENOUS	"ARTERIALIZED"	ARTERIAL
14	59.6	44.9	46.8	26.2	51.0	59.8
16	47.9	45.2	46.6	45.0	78.1	80.3
17	46.4	34.4	34.6	40.9	74.4	72.8

DISCUSSION

In patients under resting conditions, venous blood "arterialized" by the method of Goldschmidt and Light is equivalent to arterial blood in CO_2 tension. While "arterialized" blood is not identical to arterial blood in its O_2 tension, the tension does approach that of arterial blood to a reasonable degree. We feel that the degree of approximation is such that "arterialized" blood may possibly be used instead of arterial blood in many tests that involve the determination of arterial CO_2 and O_2 tensions. Experiments are now in progress in this laboratory to determine the validity of this contention.

On the basis of these experiments, and those of Goldschmidt and Light, it seems advisable to confine the sampling of "arterialized" blood to the veins on the dorsum of the hand.

SUMMARY

Blood gas tension studies on 17 patients, at rest, demonstrate that "arterialized" venous blood approximates arterial blood in its CO_2 and O_2 tension levels.

We wish to acknowledge the advice and technical assistance of Mrs. Dorothy H. Burke, Biochemist, Surgical Research Laboratory, Veterans Administration Hospital, Providence, R. I.

REFERENCES

1. Björk, V. O., Michas, P. A., and Uggla, L. G.: The Arterial O_2 Tension in Rest and During Effort as a Test of Pulmonary Function, J. THORACIC SURG. **25**: 558, 1953.
2. Björk, V. O., and Hilty, H. J.: The Change in the Arterial O_2 and CO_2 Tension During Voluntary Hyperventilation as a Test of Lung Function, J. THORACIC SURG. **27**: 541, 1954.
3. Lilienthal, J. L., Jr., and Riley, R. L.: On the Determination of Arterial Oxygen Saturations from Samples of "Capillary" Blood, J. Clin. Invest. **23**: 904, 1944.
4. Goldschmidt, S., and Light, A. B.: A Method of Obtaining From Veins Blood Similar to Arterial Blood in Gaseous Content, J. Biol. Chem. **64**: 53, 1925.
5. Filley, G., Gay, F. E., and Wright, G. W.: The Accuracy of Direct Determinations of O_2 and CO_2 Tensions in Human Blood *In Vitro*, J. Clin. Invest. **33**: 510, 1954.

MITRAL INSUFFICIENCY

Experimental Circular Suture Around the Atrioventricular Ring

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SURGICAL correction of mitral insufficiency can be possible only when the exact pathologic form of the lesion of the mitral valve has been accurately assessed. To do this with certainty is impossible by clinical means alone, it requires intracardiac exploration. With cardiotomy now a daily occurrence in the surgical world, more and more data are accumulating about the pathology of the living mitral valve, and it is quite certain that the hitherto accepted distinctions between mitral stenosis and mitral insufficiency do not cover all possible varieties of this lesion.

In a masterly analysis of the surgical and pathologic anatomy of the mitral valve, Brock[1] has stressed that, while the complex nature of the *normal* mitral valve mechanism is well understood, when dealing with the *diseased* valve, one is all too likely to concentrate on the valve cusps alone and to exclude its other components. Yet these can also be affected by the disease process. One must consider the mitral valve as an entity arising from the atrioventricular fibrous ring, with its two cusps, plus chordae tendineae *and* papillary muscles, all of which form its musculotendinous mechanism.

Basically there are six possible pathologic changes which can cause mitral incompetence.

The first two are rare, and include: (1) a hole developing in a valve cusp such as occurs with malignant endocarditis, and (2) rupture of the musculotendinous mechanism leaving the valve unsupported in ventricular systole.

These two can be reproduced experimentally.

The next four are associated with rheumatic carditis and may occur in varying degrees at the same time. There may be: (3) rigidity of the valve preventing its closure, (4) shortening of one or both cusps so that they cannot meet and overlap, (5) shortening of the musculotendinous mechanism so that the cusps cannot rise and meet, and (6) extreme dilatation of the atrioventricular ring leading secondarily to relative shortening of the cusps.

These last four forms of insufficiency cannot as yet be satisfactorily reproduced in experimental animals.

As Brock has pointed out, in practice, the incompetent mitral valve can be classified into one of three large groups:

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Group 1 represents the valves in which there is a small regurgitant jet in association with mitral stenosis and a small, tight, perhaps calcified orifice, 1 cm. or less in length. As there has been complete fusion of the commissures and therefore no loss of cusp substance, after commissurotomy the released cusps function apparently normally. This regurgitation is of little significance by itself.

Group 2 represents the valves in which there is a moderately powerful regurgitant stream occurring with an orifice of 1.5 to 2.5 cm. in length. The causative rheumatic carditis may have fused the anterior commissure, but by fibrosis and calcification held the posterior commissure widely open. Alternatively, the aortic or more usually the posterior cusp may be shortened, or again they may be tethered down by shortening of the musculotendinous mechanism to form a cone-shaped mitral opening.

Group 3 represents the valves in which there is a powerful current through a large mitral orifice at least 3 to 4 cm. in diameter. In this lesion there is no stenosis, but rather a "dehiscence of the annulus" (Black[2]), where the regurgitation is due solely to failure in normal valvular approximation.

As yet it is not possible to get reliable figures on the relative frequency of Groups 2 and 3. In a recent clinical study of 30 patients with mitral incompetence, Brigden and Leatham[3] studied 9 necropsies. The mitral ring was of normal size in 1 case only, and was dilated in the remainder. All showed thickening and deformity of the valve cusps with calcification in 2 cases. The chordae tendineae were thickened in 6 cases and in 2 of these a chorda was ruptured.

METHODS OF SURGICAL CORRECTION

In Group 1, the small jet associated with a tight mitral stenosis is an incidental finding. The treatment is correction of the mitral stenosis.

Groups 2 and 3 claim closer study. To date none of the published methods of surgical correction has been completely satisfactory in itself.

Bailey,[4] who pioneered its surgical treatment, pointed out in 1951 the relative dearth of valvular tissue in mitral insufficiency, and emphasized that surgical treatment must logically be directed along the lines of either supplying additional valve substance to bridge the aperture, or else of reducing the size of the aperture so that the available valve tissue will become adequate in amount.

To this end he at first devised two operative procedures, one in which a pedicled pericardial graft was placed across the left ventricle to *tamponade the mitral orifice from below,* and the other in which it was hoped *to narrow the valve orifice* by sewing the chordae tendineae with purse-string sutures.

Indifferent results on clinical trial, however, made him devise a third procedure[5] in which there was a direct attack on the diseased valve cusps, the attack taking one of two forms. He assumed there was predominantly one type of defect with widely separated cusps posteriorly. In this procedure the affected cusps are either suspended singly to the ventricular wall, or else the

divergent cusps are tethered together, narrowing the orifice. It is quite certain that by its nature, this method cannot relieve all types of mitral insufficiency.

Experimentally, Carter and associates[6] found that the viability of an intraventricular pericardial pedicle is doubtful even when the flap is carefully cut to preserve its normal blood supply. As an alternative method of *tamponade from below* they placed a plastic ball prosthesis in the left ventricle adjacent to the mitral valve of normal dogs as well as in dogs with surgically produced mitral insufficiency. Whereas in the normal dogs no change in the auricular pressure pattern was noted, in those with experimentally produced mitral insufficiency the prosthesis appeared to act as a "bottle baffle" giving significant pressure and wave pattern change. Johns and Blalock[7] have reported favorable results using a polyvinyl sponge prosthesis.

Instead of tamponading the dilated mitral orifice from *below,* Hayward[8] has ingeniously used the left auricular appendix and invaginated it through the mitral orifice *from above,* thereafter suturing it to the ventricular wall. Though his published immediate results were pleasing, within three months the patients had returned to their preoperative state. Autopsy showed that the tamponading left atrial appendix, subject to direct ventricular pressure, had become altered in shape, attenuated, and that the sutures had not held their position due to the constant jet from the contracting ventricle.

My own interest in active relief of mitral insufficiency was aroused when on June 8, 1953, I explored the heart of a patient who was known to have mitral insufficiency and in whom it was hoped that some degree of fusion of the commissure was present so as to allow a possible commissurotomy. Exploration of the mitral orifice through the left auricular appendix, however, showed it to be widely dilated. There was no evidence of disease of the valve cusps or musculotendinous mechanism. With each ventricular systole a strong regurgitant current of blood ballooned the left atrium. The basic lesion was in this case dilatation of the atrioventricular fibrous ring. It seemed therefore that the logical method of correcting this defect was *not* by tamponading the already dilated orifice, tethering its normal valve cusps, nor by concentrically tightening the chordae tendineae, but by passing a circular suture around the left atrioventricular fibrous ring, and, by drawing this purse string tight, thereby reestablishing the normal form and function of the ring.

Though not designed to treat mitral insufficiency, one piece of work had an important bearing on this problem. Ellison and co-workers,[9] in order to produce experimental mitral stenosis, had passed a circular suture around the left A-V ring and had then gradually tightened the ligature at a second operation 14 to 21 days later. A smooth stenosis was thus produced at the base of the mitral valve, causing no interference with closure of the mitral valve cusps. The operative mortality was minimal and the suture had been accurately placed in 95 per cent of the cases.

Knowing therefore that it was technically possible to pass a suture around the mitral ring, it seemed reasonable to hope that this technique of producing experimental mitral stenosis could be adapted to meet the requirements of

correcting mitral insufficiency, especially in the Group 3 pattern, in which a widely dilated valve orifice was present. If such a suture could be passed in human beings then the degree of tightening required to abolish the insufficiency could be gauged by atrial exploration. Further, in the Group 2 cases, if there was also an associated stenosis of one commissure, then commissurotomy could be carried out and the tightening continued to accommodate the insufficiency.

Experiments on sheep were therefore commenced on Sept. 21, 1953, and the preliminary results reported.[10]

METHOD I

The left side of the heart was exposed through the bed of the resected left fifth rib. As it was not possible to produce an insufficiency similar to that occurring with mitral rheumatic disease, it was decided to pass the suture and observe its effect on the normal heart, by examining the heart three months after operation, and assessing the narrowing of the ring.

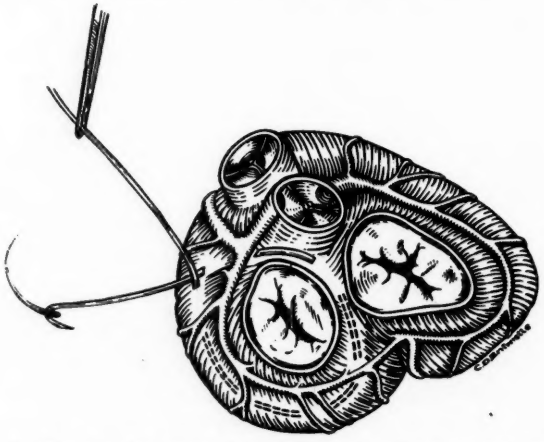

Fig. 1.—Method I. The circular suture lay below the coronary artery, passing under its branches.

The first step was to dissect free the main left coronary artery as it arose from the aorta, to define its course in the transverse sinus, and to isolate the origin of its two major divisions. When these had been isolated, a double floss-silk suture was inserted on a No. 8 round-bodied needle. Floss silk is strong, has been well tried for repair of direct inguinal hernia (Maingot[11]) and, when lubricated with sterile paraffin and inserted doubled, is sufficiently bulky to seal any needle hole.

In the first series of experiments, the suture commenced on the left side of the ventricle just below the main division of the left coronary artery, and, by under-running all obvious branches, continued to the inferior interventricular sulcus where left and right coronary arteries anastomose (Fig. 1). At this point, the suture was passed through the heart along the interven-

tricular septum to emerge in the transverse sinus just behind the origin of the aorta. It was then brought laterally to just above the left coronary artery, but then was passed beneath it and so back to the starting point. The slack being taken up, the suture was tightened by 1 cm. and tied. The pericardium was resutured, and the chest wall closed over water-seal drainage. Two weeks after operation, the animals were sent to graze at Invermay Research Farm.

RESULTS OF METHOD I

Immediate Result.—This first experiment of placing the circular suture below the left coronary artery was technically developed and performed on 18 sheep, 11 of whom made an uninterrupted recovery from operation and 7 died from operative causes. Four of these deaths were due to anesthetic difficulties, namely, inhalation of postoperative vomit (3) and lung fistula (1). The remaining 3 developed acute coronary infarction and died within an hour of the operation.

Comment.—This coronary infarction clearly showed that, although 11 recovered from operation, the placing of the suture below the left coronary artery was wrong in principle. The suture should lie entirely above the left coronary artery.

Late Results.—Three months after operation the animals were killed and their hearts sectioned to show the mitral valve. The story of the development of the technique is shown in Figs. 2, 3, and 4. The normal sheeps' heart has a marginal artery passing down the left border of the left ventricle. In the earliest experiments, when one was chary about raising the heart from the pericardial cavity, this was erroneously taken as the most posterior point at which the circular suture should be placed in order to lie opposite the interventricular septum. In this animal, the transcardiac suture was later shown to lie to the left of the mitral orifice (Fig. 2).

Fig. 3 shows that the transcardiac suture had entered the heart midway between the left marginal artery and inferior interventricular groove. As the suture, in fact, lay directly across the mitral orifice, fibrin forming on it had produced an obstruction to the mitral orifice. While being put to pasture two weeks after the operations, the sheep, chased by an untrained pup, ran wildly around the sheep yard and died within five minutes from acute pulmonary edema.

Fig. 4 shows that the sutures had just missed passing through the interventricular septum.

Comment.—These there results conclusively showed the necessity for making the transcardiac suture enter at the inferior interventricular groove and pass on into the interventricular septum. Having thus established these facts, eleven subsequent transcardiac sutures were placed correctly in the interventricular septum. In none did cardiac irregularity arise from possible interference with the A-V bundle or other branch of the conducting mechanism within the heart. Where the suture has been accurately placed, there develops a thickening of the A-V ring which nevertheless allows normal functioning of the mitral valve cusps and chordae tendineae.

Fig. 2.

Fig. 3.

Fig. 4.

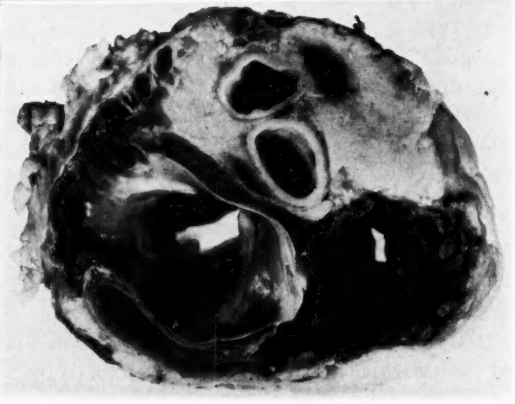

Fig. 2.—Initial attempt. The transcardiac suture crossed to the left of the mitral orifice.

Fig. 3.—Second attempt. With the suture over the mitral orifice, fibrin forming on it caused mitral obstruction within 2 weeks.

Fig. 4.—Third attempt. Suture lying just to left of interventricular septum.

METHOD II

Using the same approach to the heart as before, the atrioventricular groove was displayed by lifting the auricular appendage with an auricular clamp. The coronary sinus was carefully dissected free of cardiac fat throughout its course in the A-V sulcus. The left coronary artery was also dissected from its origin across the transverse sinus to its two major branches, from which point the posterior one was followed. Vertical sections through the heart show that the fibrous left A-V ring lies in the depths of this sulcus.

The suture was carefully inserted to lie at the base of the fibrous ring (Fig. 5). It took at least three bites of the needle to place the suture at the side and back of the fibrous ring.

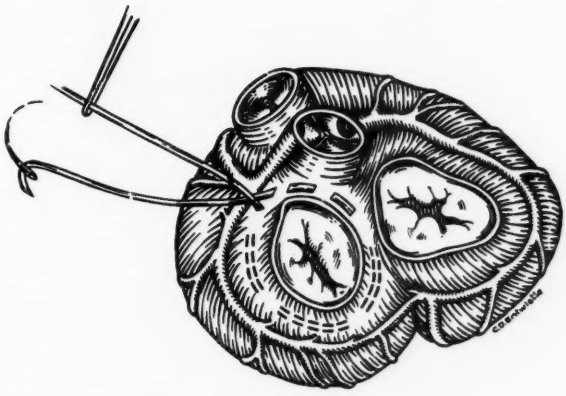

Fig. 5.—Method II. The circular suture lies above the coronary artery and at the base of the fibrous ring.

The transcardiac suture was placed as in Method I. It was surprising how relatively easily the atria can be compressed from before back and the needle passed forward on to the left index finger waiting to receive its sharp point in the transverse sinus. The needle was then caught between index and middle fingers and guided out of the transverse sinus. Transient ooze was controlled by digital pressure. Finally, after the transverse sinus was opened by retraction of the left auricle and pulmonary artery, the last stitch was inserted beneath the coronary sinus to the starting point. The suture was tightened as before.

RESULTS OF METHOD II

Immediate Results.—These were encouraging as in 9 experiments all animals survived.

Late Results.—Eight of the 9 sheep were killed 2 to 3 months after operation and their hearts examined. One sheep is being kept alive for over a year to

determine the long-term result. The heart showed a firm, narrowed left A-V ring with normal functioning valve cusps. In several the diameter of the ring had been reduced to half its normal width (Figs. 6 and 7).

Fig. 6.

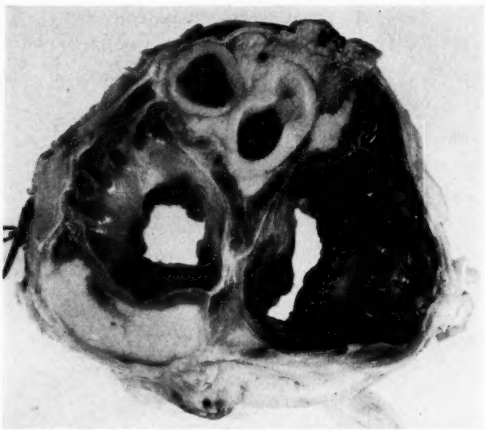

Fig. 7.

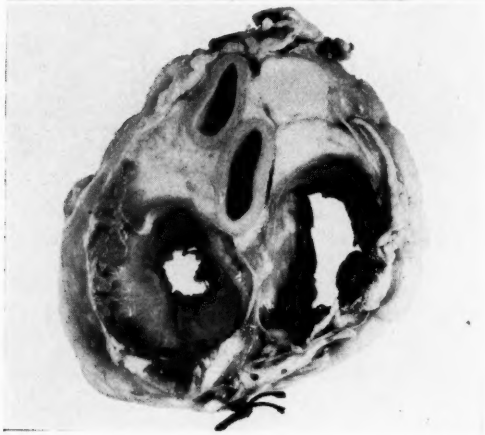

Fig. 6.—Method II. Circular constriction of left A-V ring without disturbing cusp function.

Fig. 7.—Result similar to Fig. 6.

The atria were cut away to show the upper surface of the mitral and tricuspid orifices. Depending on the degree of tightening of the circular suture before ligation, so the diameter of the left A-V ring varied, but in several it was reduced to almost half its normal diameter. It was very firm and, as histological examination confirmed, more fibrous than normal (Figs. 8, 9, and 10). The valve cusps and chordae tendineae had continued to function normally.

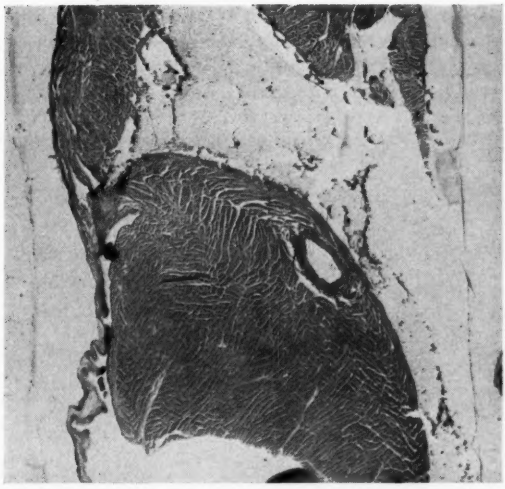

Fig. 8.—Normal histological appearance of the left A-V ring (Van Giesen).

Fig. 9.—Fibrous reaction around floss-silk suture (Van Giesen) at region of fibrous ring.

COMMENT

These results showed therefore: (1) that the left A-V ring can be effectively narrowed by passing a floss-silk suture around it and tightening the suture, (2) that the mitral valve cusps continue to function adequately under such circumstances, and (3) that the fibrous A-V ring is considerably thickened by the presence of this suture.

Fig. 10.—Further example similar to Fig. 9.

SUMMARY AND CONCLUSIONS

The fibrous ring supporting the mitral valve can be effectively narrowed by passing a double floss-silk circular suture around it. In the initial 18 experiments, where the suture lay below the left coronary artery, although 11 animals recovered from operation, 3 died from acute coronary occlusion. In 9 subsequent experiments, where the suture lay above the left coronary artery, all animals recovered. The resulting narrowing and thickening of the A-V ring did not interfere with the action of the mitral valve cusps and chordae tendineae.

It is concluded that this method may well prove to have a real place in the surgical correction of mitral insufficiency, especially in patients with a widely dilated mitral valve ring. Cardiotomy would be the first step to assess the pathologic type of mitral insufficiency. Thereafter, the circular suture would be inserted, and the degree of tightening judged by intracardiac palpation.

REFERENCES

1. Brock, R. C.: The Surgical and Pathological Anatomy of the Mitral Valve, Brit. Heart J. **14:** 489, 1952.
2. Black, H.: In discussion: Carter, M. G., Gould, J. M., and Mann, B. F.[6]
3. Brigden, W., and Leatham, A.: Mitral Incompetence, Brit. Heart J. **15:** 55, 1953.
4. Bailey, C. P., Lacy, M. M., and Harris, J. S. C.: The Surgical Treatment of Acquired Heart Disease, S. Clin. North America **31:** 1821, 1951.
5. Bailey, C. P., Bolton, H. E., and Redondo-Ramirez, H. P.: Surgery of the Mitral Valve, S. Clin. North America **32:** 1807, 1952.
6. Carter, M. G., Gould, J. M., and Mann, B. F.: Surgical Treatment of Mitral Insufficiency. An Experimental Study, J. THORACIC SURG. **26:** 574, 1953.
7. Johns, T. M. P., and Blalock, A.: Mitral Insufficiency. The Experimental Use of a Mobile Polyvinyl Sponge Prosthesis, Ann. Surg. **140:** 335, 1954.
8. Hayward, J.: A New Operation for Mitral Regurgitation, Australian & New Zealand J. Surg. **23:** 257, 1954.
9. Ellison, R. G., Major, R. C., Pickering, R. W., and Hamilton, W. F.: Technique of Producing Mitral Stenosis of Controlled Degree, J. THORACIC SURG. **24:** 154, 1952.
10. Borrie, J.: Mitral Insufficiency: Circular Suture Tightening the A-V Ring, Proc. Univ. Otago Med. Sch. **32:** 24, 1954.
11. Maingot, R. H.: Floss Silk Lattice Repair for Direct Inguinal Hernia, J. Internat. Coll. Surg. **8:** 1, 1945.

BLOODY PLEURAL EFFUSION CAUSED BY A BRONCHOGENIC CYST

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THE common clinical teaching that a bloody pleural effusion is secondary to carcinoma or tuberculosis is usually valid. Recently, however, we have had under our care an unusual case of bloody pleural effusion secondary to a bronchogenic cyst, which forms the basis of this report.

CASE REPORT

B. J. (B. C. H., No. 1505272), a 20-year-old single white woman, entered the Boston City Hospital for the first time on March 30, 1954, with the chief complaint of chest pain and shortness of breath of four days' duration. She stated that, one month prior to admission, she had developed a cold with a cough productive of brownish-black sputum, which continued up to the time of admission. She was able to continue work as a sales clerk in a department store, although she did not feel well. Her appetite remained good. There was no weight loss. There had been occasional mild night sweats, but the patient felt that these usually followed the taking of aspirin.

Four days prior to admission, while at work she was suddenly seized with a substernal pain described as sharp and stabbing and which was aggravated by breathing and coughing. After a short rest, she was somewhat relieved and went back to work. It was noted that with the onset of sharp pain, there was associated shortness of breath and this had gradually increased until the time of admission to the hospital. The location of the pain changed gradually and was felt most acutely in the left upper anterior chest, remaining pleuritic in nature. There was no history of contact with tuberculosis. She had had pneumonia at the age of 6, at which time her mother stated that she was "run down and thin." Questioning revealed that she smoked one package of cigarettes per day. Leg cramps had been noted occasionally, but without calf tenderness. There was no history of chills and fever, or previous heart disease.

Physical examination revealed a fairly well-developed, well-nourished, young white woman, showing marked dyspnea. There was mild convergent strabismus of the right eye. The skin of the face revealed extensive acne. The teeth were markedly carious. Examination of the chest revealed signs of pleural effusion up to the apex on the left with marked mediastinal and tracheal shift to the right. The heart sounds were heard best to the right of the sternum. The remainder of the physical examination was essentially negative.

Laboratory.—The urinalysis was negative. The hematocrit was 34 per cent. The corrected sedimentation rate was 22 mm. per hour. The white blood count was 13,700 cells, showing 13 per cent monocytes, 12 per cent lymphocytes, and 75 per cent polymorphonuclear leukocytes. Of the latter, 50 per cent were young forms. X-rays revealed a homogeneous density of the entire left chest (Fig. 1). A chest tap produced 1,100 c.c. of bloody fluid.

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The specific gravity of this fluid was 1.022; the total protein was 3 Gm. Cell count of the aspirated fluid revealed 25,000 red blood cells per cubic millimeter and 20,000 white blood cells per cubic millimeter. Material sent for guinea pig examination, cytologic examination, and cell block revealed negative results in all cases. Ziehl-Neelsen stain of fluid sediment was negative for acid-fast bacilli.

Course.—Oxygen and sedatives were administered for severe dyspnea and the patient was put on tuberculosis precautions. Seven more thoracenteses were performed and 150 to 800 c.c. of bloody fluid was aspirated each time. All of the fluid recovered was similar to that obtained in the first tap. X-rays continued to show an extensive fluid collection and thickened pleurae of the entire left chest. Fifteen sputum examinations, five gastric cultures and skin tests to second strength purified protein derivative were negative for tuberculosis. Chest fluoroscopy on April 13, 1954, revealed only mediastinal shift and demonstrated diminished motion of the left diaphragm. An electrocardiogram revealed sinoauricular tachycardia. Bronchoscopic examination on April 16, 1954, under topical anesthesia revealed marked narrowing of the distal left main-stem bronchus without any intrinsic pathology being noted. In spite of all studies, the consensus was that this patient had either tuberculosis or tumor. Diagnostic pneumothorax and thoracoscopy were recommended. These were carried out on April 22, 1954. Direct observation revealed only fibrin and bloody fluid, and a pleural biopsy showed only "fibrin and pleuritis." Since the patient was making no clinical progress and all other diagnostic measures had been exhausted, exploratory thoracotomy was resorted to on May 3, 1954.

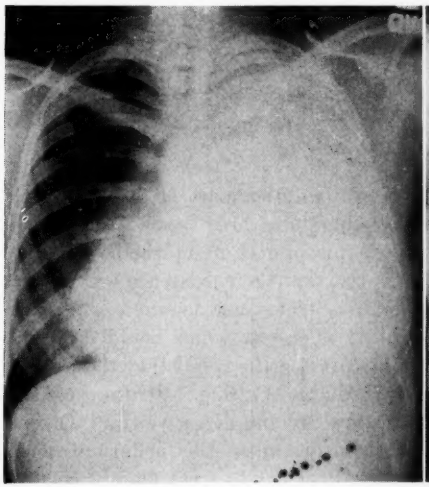

Fig. 1.

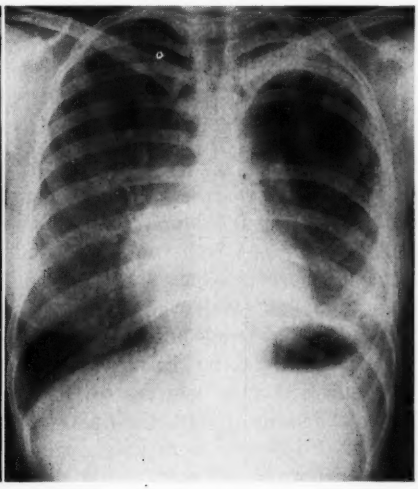

Fig. 2.

Fig. 1.—Shows massive pleural effusion on the left with accompanying mediastinal shift.
Fig. 2.—Postoperative x-ray revealing complete expansion of the left lung with a small apical pleural cap.

Through the bed of the sixth rib, the pleural cavity was entered and approximately 1.5 L. of bloody fluid was evacuated. The walls of the left hemithorax were covered everywhere by shaggy, fibrinous exudate. On the mediastinal aspect, the lung was compressed to approximately one-fourth of its normal size. Once this cortex was entered by sharp dissection over the lung, it peeled quite easily and was approximately 3 mm. in thickness. As it was removed from the lower surface of the lung, it was realized that a large cystic structure also was being uncovered. This structure measured approximately 20 cm. in diameter and

was bounded below by the diaphragm, medially by the posterior mediastinum, and superiorly by the inferior surface of the lung, which it pushed upward. The lung was completely atelectatic. The cystic structure was completely separated from the lung and diaphragm and was found to be attached by a 2 cm. stalk to the lower esophageal wall. This stalk appeared to be composed of muscle fibers, vessels, and nerves. Rupture of the cyst during its removal showed it to contain approximately 700 c.c. of greenish-gray, mucoid material. The palpating finger within the cyst was easily able to feel the Levin tube within the esophagus, with only mucosa intervening between the finger and the tube. The stalk was then amputated and the defect in the esophageal musculature was closed with interrupted silk sutures. Decortication of all visceral and parietal surfaces was then carefully done and the lung expanded. It was noted that even with pressure, the expanded lung would not completely fill the hemithorax. Two drainage tubes were placed in the chest and the wound closed.

Postoperatively, the drainage tubes were placed on suction of 10 cm. of water negative pressure for five days. Since these became obstructed, they were removed and a new tube was placed in the upper second intercostal space anteriorly, and again this was placed on suction. Thus, the pleural cavity was kept on suction for one week following operation. With the removal of the intrathoracic tubes, resort was made to daily tapping and instillation of streptokinase-streptodornase solution for three days. In spite of all these measures, plus deep-breathing exercises, the lung never expanded to fill the hemithorax completely. The aspirated material rmained sterile and the patient was discharged to be followed carefully, lest empyema develop.

Postoperative follow-up five months following operation revealed a completely expanded lung with a pleural cap (Fig. 2). Fluoroscopy showed only slight motion of the diaphragm on the left.

The pathological diagnosis on the resected cystic specimen was that of "bronchogenic cyst."

DISCUSSION

The theories concerning embryogenesis of enterogenous or bronchogenic cysts of the mediastinum can be divided roughly into three types. Lewis and Thyng[1] proposed embryonic diverticula or outcroppings of epithelial nodules capable of producing gastric mucosa. Keith[2] felt that their existence was caused by a failure of coalescence of the vacuoles in a transverse manner, when the alimentary tube was in a solid state, resulting in a cyst which could or could not be attached to it. Black and Benjamin[3] expressed the belief that these cysts were intrathoracic vestiges of the vitelline duct. Calling attention to the common genesis of epithelial lining of the respiratory and digestive tract, Olken[4] indicated difficulty in making a diagnosis as to origin on this criterion alone. This writer pointed out further that all mediastinal cysts were closely related embryologically; however, he added, the esophageal cysts seemed to form a rather distinct group. They are almost invariably found in an adult, situated in the lower half of the esophagus, usually between the muscle layers of the esophageal wall, and the epithelium is composed of ciliated cuboidal cells with occasional foci of flattened epithelial cells. Histologically, these cysts resemble most closely the group described as of bronchogenic origin, except that they contain no cartilage. It would be expected that these cysts would cause symptoms secondary to pressure on the contiguous mediastinal structure. Dyspnea, cough, cyanosis, pleurisy, dysphagia can all be produced in some degree in this manner. However, the more alarming clinical syndromes are due to another

feature of these cysts. Because of their common tendency to contain gastric secretory epithelium, peptic ulceration, secondary inflammation, and perforation into a bronchus or pleura may cause a patient and the physician justifiable alarm.[5-7]

It is generally agreed that the only treatment which can be recommended is removal of these cysts as soon as they are discovered in order to avoid secondary compressive or inflammatory complications.

SUMMARY

A case of bloody pleural effusion secondary to mediastinal compression by, or rupture of, a bronchogenic cyst is reported.

Although rare, it is felt that this is an important diagnostic possibility to bear in mind when confronted with the problem of unexplained bloody pleural effusion.

REFERENCES

1. Lewis, F. T., and Thyng, F. W.: The Regular Occurrence of Intestinal Diverticula in Embryos of the Pig, Rabbit and Man, Am. J. Anat. **7**: 505-519, 1907-1908.
2. Keith, A.: Human Embryology and Morphology, Baltimore, 1933, William Wood and Co.
3. Black, R. A., and Benjamin, E. L.: Enterogenous Abnormalities; Cysts and Diverticula, Am. J. Dis. Child. **51**: 1126-1137, 1936.
4. Olken, H. G.: Congenital Gastro-enteric Cysts of the Mediastinum; a Review and Report of a Case, Am. J. Path. **20**: 996-1010, 1944.
5. Davis, E. W., and Salin, D.: Intrathoracic Gastric Cysts, J. A. M. A. **135**: 218-221, 1947.
6. Gross, R. E.: The Surgery of Infancy and Childhood. Its Principles and Techniques, Philadelphia, 1953, W. B. Saunders Co.
7. Lindskog, G. E., and Liebow, A. A.: Thoracic Surgery and Related Pathology, New York, 1953, Appleton-Century-Crofts, Inc.

THE ONE-BREATH OXYGEN TEST ADAPTED FOR USE WITH DIFFERENTIAL BRONCHOSPIROMETRY

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THE one-breath oxygen test described by Comroe and Fowler[1] has proved a valuable method for measuring uneven alveolar ventilation. We have modified the described test by discarding the flowmeter to obtain the volume curve. The Lilly flowmeter is expensive, the fine mesh is easily affected by condensation of water vapor, the unevenness of the curves suggests the presence of artifacts, and the calculation of volume is laborious. To measure the volume of expired air, we use a 7 L. well-balanced respirometer. Over the shaft of the pulley is attached a potentiometer of 360 degrees, low torque and 10,000 ohms*, connected to a Sanborn Poly-Viso recorder. The contact voltage is provided by a 1.5 volt battery and rheostat. The curve for volume of gas expired is linear in the range and rates of flow used for the test. Simultaneous recording of flow and the curve from the nitrogen meter allows one to calculate easily the percentage rise of the nitrogen curve. The points 750 c.c. and 1,250 c.c. are generally used, as recommended by Comroe, but the occasional patient is unable to expire in this range. When this occurs the same increment may be used at lower points provided they fall on the alveolar plateau.

Fig. 1 shows a typical abnormal test taken from a patient with moderate emphysema. The upper portion of the figure on the left shows the standardization of 0 per cent to 80 per cent nitrogen. On the right is the nitrogen curve showing the inspiration of 100 per cent oxygen, the expiration of dead space air, the rising curve representing the mixture of dead space air and alveolar air and the so-called alveolar plateau on which is calculated the 8 per cent rise of nitrogen in 500 c.c. of alveolar air. The dotted line indicates a normal curve for comparison. The lower portion of the figure shows the gas flow curve from which perpendicular lines are drawn at 750 c.c. and 1,250 c.c. of expired air. It is important to have the patient expire evenly and slowly to compensate for the slight lag in the nitrogen meter.

We have used the one-breath oxygen test in screening over 100 patients before pulmonary resection. There is good correlation between this test, a prolonged timed vital capacity, and reduced maximum breathing capacity with spirographic curve elevated to the hyperinflation level. All patients with elevated ratio of residual air to total capacity and increased index of intrapulmonary mixing had grossly abnormal one-breath oxygen tests.

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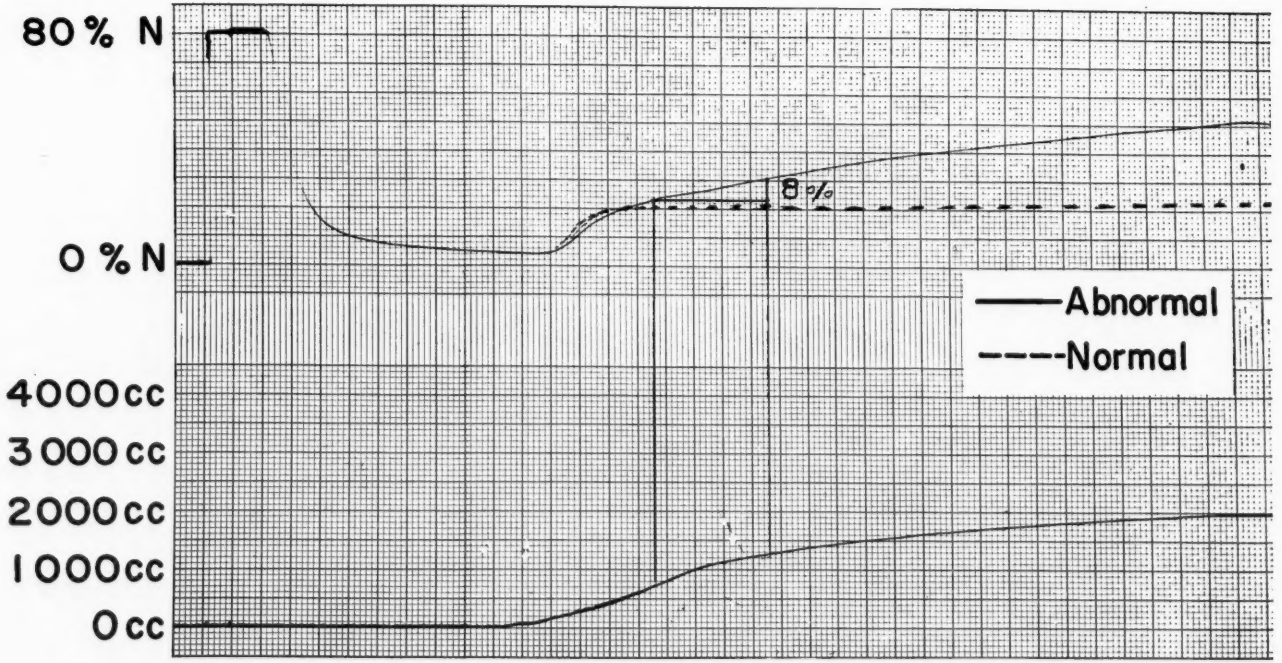

Fig. 1.—One-breath oxygen test which shows nitrogen curve on upper graph and expiratory volume curve on lower graph.

Over 50 per cent of our patients undergo pulmonary resection for residual tuberculous lesions. Many of these receive lobectomies without thoracoplasty or plombage. We have undertaken a long-term study to determine whether the remaining overexpanded lung develops emphysema. We have scanty pathologic evidence that this occurs, but the rapidity, extent, and frequency of these changes are not known. A change from a normal one-breath oxygen test before pulmonary surgery to an abnormal curve postoperatively might indicate the development of poor intrapulmonary gas distribution and suggest the possibility of some emphysema. It then becomes important to determine which lung is involved and to measure the degree of abnormality.

Differential bronchospirometry will provide comparison between the two lungs for ventilation, vital capacity, and oxygen uptake. The seven-minute nitrogen washout test of Darling[2] is not practical for bronchospirometry because of the length of time necessary to complete the tests bilaterally. A patient will rarely tolerate bronchospirometry for more than a few minutes and hyperventilation occurs in many of them during the procedure. The one-breath oxygen test is ideally suited to bronchospirometry. The test is completed quickly without further discomfort to the patient. Nitrogen curves of the expired gas from each lung are obtained which allow direct comparison and analysis.

METHOD

Bronchospirometry is performed according to the standard technique using a Gebauer or Carlen double lumen catheter.[3] The tubes of the catheter are connected to a Collins bronchospirometry assembly with two respirometers. On the pulley shaft of one of these is fitted a potentiometer connected to a Sanborn Poly-Viso recorder as described previously. The needle for the nitrogen sampler is inserted into the connecting couple just above the end of the catheter tube. The tubing leading to the Waters-Conley nitrogen meter is 12 inches in length. The output from the amplifier is connected to another channel on the Poly-Viso. The potentiometer and nitrogen meter are carefully standardized during the time the patient is receiving the local anesthesia in preparation for the insertion of the catheter. After the catheter is in place the three-minute run on room air is made, during which ventilation and oxygen uptake are obtained. Then the catheter is disconnected from the respirometer assembly. The respirometer tubing is interchanged so that oxygen may be breathed out of one respirometer and the expired gas is collected in the other respirometer with the potentiometer attachment. The inspiratory tubing on the latter respirometer is stoppered with a cork to prevent gas leakage. When all is in readiness and the respirometer bell is filled with 99.4 per cent oxygen, the patient is asked to take a deep breath of room air and to exhale completely. He is then connected with the oxygen circuit and he is again asked to take a deep breath, at which time the Sanborn recorder is started. He then exhales slowly and evenly into the second respirometer while the nitrogen curve and expiratory volume are recorded. The same procedure is repeated for the opposite side by merely interchanging the catheter tubes. Usually one recording from each side suffices. Occasionally the patient may cough during the

procedure, in which case the test may be repeated after a minute or two. When satisfactory curves have been obtained from both sides, the nitrogen meter sampling needle is withdrawn, the tubing is reconnected to the broncho-spirometry assembly and the ventilation, oxygen uptake, and vital capacity are obtained from both sides while the patient breathes pure oxygen. The procedure is then completed by withdrawing the catheter and asking the patient to cough up any accummulated secretions. The patient is instructed not to take liquids or food for a period of two hours in order to allow the local anesthetic to dissipate.

RESULTS

The potentialities of this test may best be illustrated by selection of several typical examples.

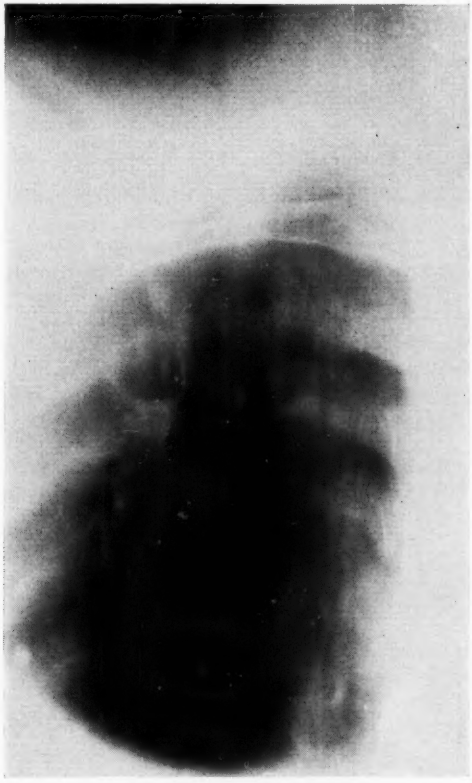

Fig. 2.—Planigram of right apex of Patient L. T. A., which shows several large emphysematous blebs.

Case 1.—L. T. A., a 32-year-old white man, was admitted to the Madison Veterans Administration Hospital on Aug. 23, 1954. A mobile x-ray unit in November, 1953, made a report of pulmonary tuberculosis, but the patient was not hospitalized until January, 1954. The sputum was found to be positive for acid-fast bacilli and he was started on

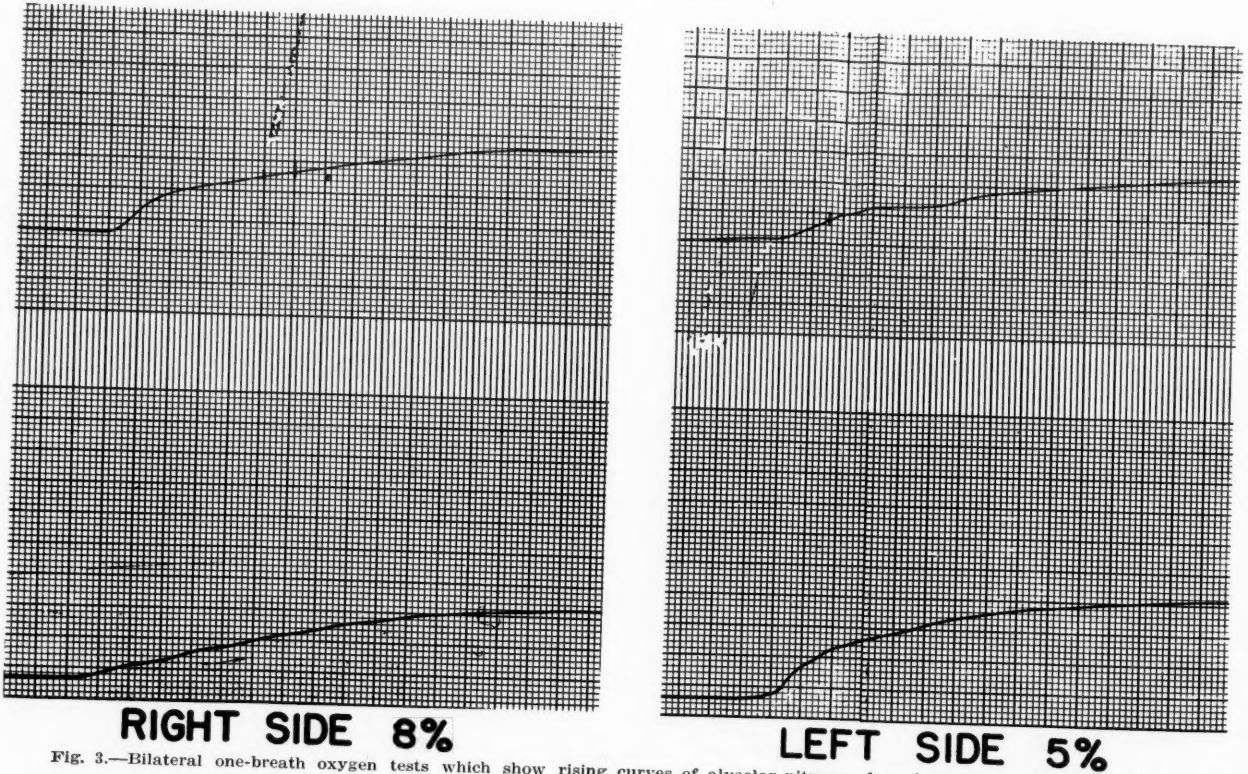

Fig. 3.—Bilateral one-breath oxygen tests which show rising curves of alveolar nitrogen from both lungs of Patient L. T. A.

antimicrobial therapy in April, 1954. At this hospital he continued with streptomycin, 1 Gm., twice weekly, and para-aminosalicylic acid, 12 Gm. daily. X-rays showed bilateral apical infiltration with possible blebs or cavity on the right as seen on planigrams (Fig. 2). He was scheduled for resection of the right upper lobe on Oct. 25, 1954. Pulmonary function studies were as follows: fluoroscopy showed lung fields as illustrated on x-ray, diaphragm moved well bilaterally. Vital capacity was 4,866 c.c. or 121 per cent of normal. Three-second timed vital capacity was 90 per cent (normal 97 per cent). Maximum breathing capacity was 107 L. or 84 per cent of normal. Spirogram showed prolonged expiration on vital capacity test and maximum breathing capacity performed at hyperinflation level. Ratio of residual air/total capacity $\times$ 100 = 27 per cent (normal less than 35 per cent). Index of intrapulmonary mixing was 5 per cent (normal less than 2.5 per cent). One-breath oxygen test showed a rising curve of alveolar nitrogen 5 per cent in 500 c.c. (normal less than 1.5 per cent), with a further rise at the end of expiration of 10 per cent. Bronchospirometry: right side—ventilation 47 per cent, vital capacity 43 per cent, O_2 uptake on 100 per cent O_2 55 per cent. The one-breath nitrogen curve on the right showed an 8 per cent rise in 500 c.c., and on the left 5 per cent rise in 500 c.c. (Fig. 3). On Nov. 19, 1954, the patient underwent resection of the right upper lobe and wedge resection of emphysematous blebs, anterior margin of the middle lobe. The pathologic sections showed emphysema and fibrocaseous tuberculosis.

Comment.—This patient had bilateral apical tuberculosis with the most marked involvement on the right. Pulmonary function studies showed evidence of air trapping and poor gas distribution. Differential bronchospirometry using the one-breath oxygen test demonstrated that the poor mixing was worse on the right. Emphysema and blebs were found at operation.

CASE 2.—M. T., 28-year-old Negro man, was admitted to this hospital Aug. 19, 1954. He developed tuberculosis of the right hip in 1945 and x-rays revealed far advanced pulmonary tuberculosis. He was hospitalized, sputum was positive, and he had received antimicrobial treatment almost continuously from 1948 to the present time. While in this hospital, he received streptomycin 1 Gm., twice weekly, and Isoniazid, 100 mg., three times daily, X-rays of the chest revealed bilateral disease with the major involvement in the right upper lobe and middle lobe. The Surgical Board recommended resection on the right side on Nov. 11, 1954, for extensive bronchiectasis in the right upper and middle lobes, as revealed by bronchograms (Fig. 4). The pulmonary function studies were as follows: fluoroscopy showed an elevated right diaphragm which moved only half the excursion of the left. On inspiration, the mediastinum shifted toward the right. Vital capacity was 3,033 c.c., or 68 per cent of normal. Three-second timed vital capacity was 84 per cent. Maximum breathing capacity was 74 L., or 53 per cent of normal. Spirogram showed prolonged expiration on vital capacity test and maximum breathing capacity performed at hyperinflation level. One-breath oxygen test showed a rising curve of alveolar nitrogen to be 6 per cent in 500 c.c. Bronchospirometry on the right showed ventilation 29 per cent, vital capacity 33 per cent, O_2 uptake on 100 per cent O_2 22 per cent. The one-breath oxygen test showed marked rise of the alveolar nitrogen curve on the right side 16 per cent in 300 c.c. and 3 per cent in 500 c.c. on the left (Fig. 5). On Nov. 28, 1954, the right upper and middle lobes were removed. These lobes were fibrotic and retracted. The lower lobe inflated well and compensated for the atelectatic lobes. Pathologic sections showed extensive tuberculous fibrosis and bronchiectasis.

Comment.—This patient had long-standing pulmonary disease with destruction of the right upper and middle lobes and bronchiectasis. Pulmonary function studies showed reduced pulmonary ventilation, evidence of air trapping, and poor distribution of gas. Differential bronchospirometry showed reduced function on the right and severely impaired mixing of gas. Bronchiectasis of the right upper and middle lobes and overdistention of the remaining lower lobe probably are responsible for the latter abnormality.

CASE 3.—S. H., a 58-year-old white man, was admitted to this hospital on Oct. 7, 1954, as a transfer. He entered another hospital in May, 1949, for an abscess of the

buttock. X-ray of the chest revealed moderately advanced pulmonary tuberculosis. The sputum was positive for acid-fast bacilli. From November, 1949, until the present time he had received several courses of antimicrobial therapy. In 1952, because of positive secretion, he had a series of operations performed at another hospital: Sept. 18, 1952, right upper and middle lobectomies; Oct. 13, 1952, thoracotomy for closure of bronchopleural fistula; Nov. 18, 1952, thoracotomy and drainage of empyema; Oct. 13, 1953, thoracoplasty, 7 ribs, and paraffin pack. Subsequently the paraffin sloughed via the tracheobronchial tree and through the thoracotomy wound. On July 6, 1954, an incision and drainage of a chest wall abscess was performed. The last positive secretions were found in May, 1953. On admission to this hospital he was continued on Isoniazid, 100

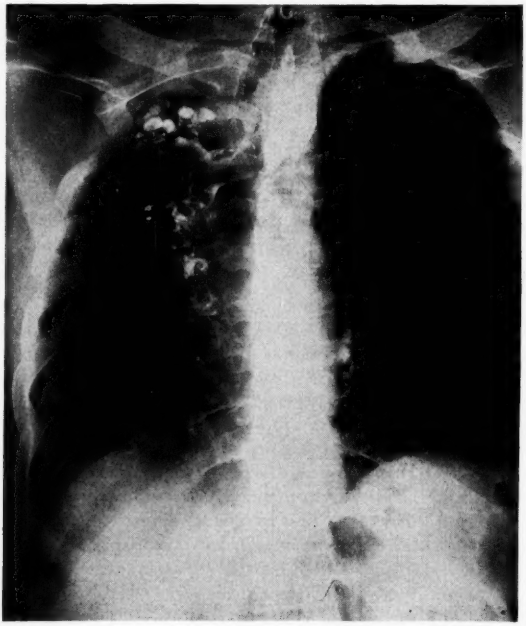

Fig. 4.—Bronchogram of Patient M. T., which demonstrates extensive bronchiectasis of right upper lobe.

mg. three times a day, and para-aminosalicyclic acid, 12 Gm. daily, but in view of possible further surgery, he was begun later on Viomycin, 2 Gm., twice weekly, and Isoniazid, 100 mg. three times daily. Because of a persistent air pocket in the right apical region (Fig. 6) and some bronchiectasis of the remaining right lower lobe, the Surgical Board recommended the completion of a right pneumonectomy. Pulmonary function studies were as follows: fluoroscopy showed a right thoracoplasty. The right diaphragm was elevated and adherent to the lateral chest wall. There was some movement of the medial portion. The left diaphragm moved well. Vital capacity was 1,971 c.c., or 55 per cent of normal. Three-second timed vital capacity was 78 per cent. Maximum breathing capacity was 54 L., or 57 per cent of normal. Spirograms showed prolonged expiration on vital capacity test and maximum breathing capacity performed at hyperinflation level. One-breath oxygen test revealed a rising curve of alveolar nitrogen to be 8 per cent in 500 c.c. Oximeter studies showed the peripheral blood to be 95 per cent saturated with oxygen while breathing room air. When the saturation was lowered to 80 per cent by rebreathing from a respirometer gradually depleted in oxygen, it took 75 seconds to return to 90 per cent

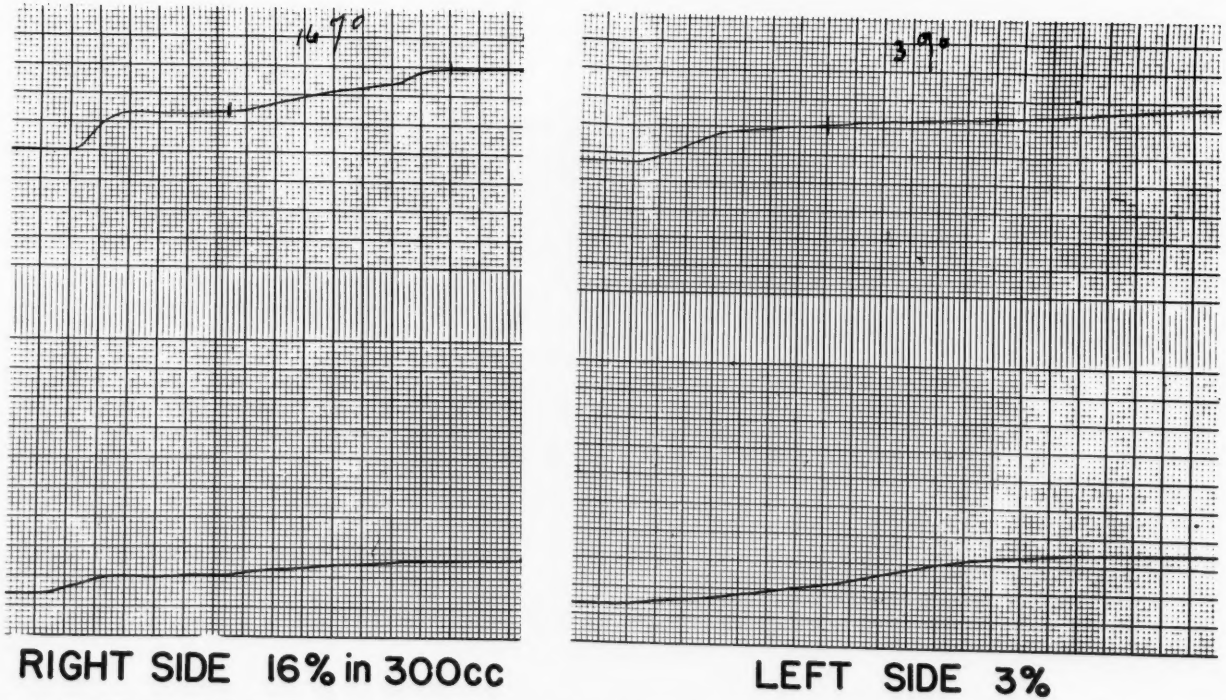

Fig. 5.—Bilateral one-breath oxygen tests from Patient M. T., which show rising, uneven curve of alveolar nitrogen from the right lung.

saturation when allowed to breathe room air again (normal less than 24 seconds).[4] Differential bronchospirometry showed ventilation on the right side of 36 per cent, vital capacity 22 per cent, and O_2 uptake 59 per cent. The one-breath oxygen test showed a rising curve on the left with 8 per cent in 500 c.c. (Fig. 7) but only 1 per cent in 200 c.c. on the right side.

Comment.—This patient was scheduled for completion of right pneumonectomy. Because of slight dyspnea on ward activity, his alert physician requested careful pulmonary physiologic appraisal. The finding of good oxygen uptake on the right was unexpected. The poor function on the left was explained by the emphysema and uneven gas distribution which was demonstrated by differential bronchospirometry. Surgery was deferred.

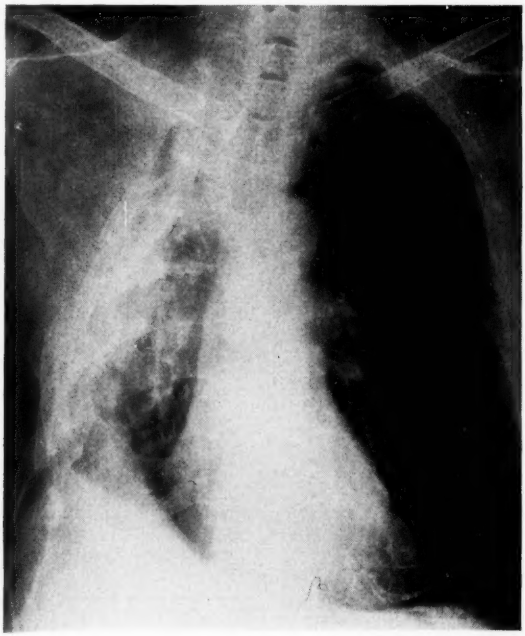

Fig. 6.—Chest x-ray of Patient S. H., which shows right thoracoplasty and extra and intrapleural air pockets at right apex.

DISCUSSION

The alveolar nitrogen curves obtained from each lung separately during differential bronchospirometry allow one to compare the gas distribution of the right and left lungs. The importance of making such observations is well illustrated by the three cases presented. We believe this method will be helpful in determining changes in the overexpanded lung following lobectomy without thoracoplasty or plombage.

It will be noted that the dead space is considerably reduced by the catheter so that the alveolar plateau is reached after expiration of about 100 c.c. of air. This allows one to take points at less than 750 c.c. and 1,250 c.c. which would not be available on lungs severely compromised by disease. When

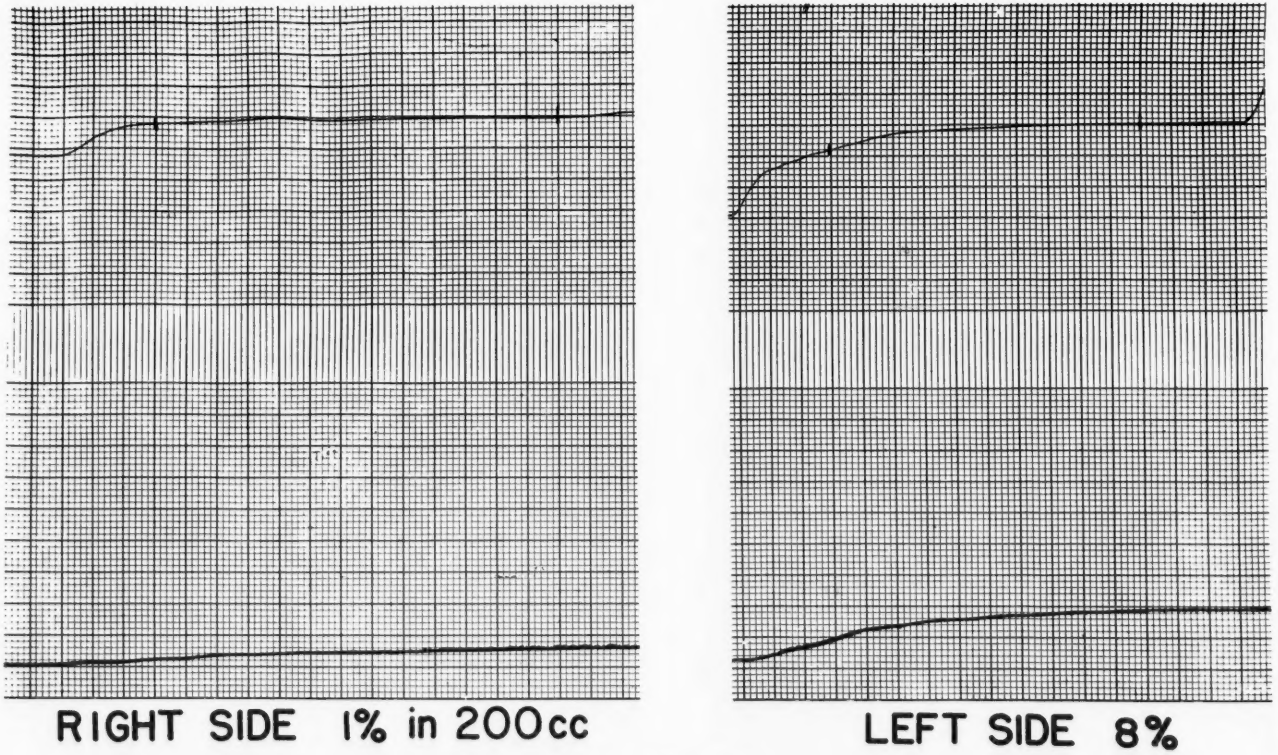

Fig. 7.—Bilateral one-breath oxygen tests on Patient S. H., which reveal unexpected poor gas distribution in left lung.

less than 500 c.c. is available on the alveolar plateau, smaller amounts may be used. The configuration of the curves, however, from the two sides provides useful evidence of uneven gas distribution, even without making the calculations.

SUMMARY

1. The one-breath oxygen test of Comroe is a sensitive indicator of alveolar gas distribution.

2. This test has been adapted to differential bronchospirometry.

3. Three case histories are presented which illustrate the value of obtaining bilateral alveolar nitrogen curves after one breath of oxygen.

REFERENCES

1. Comroe, J. H., Jr., and Fowler, W. S.: Lung Function Studies: VI. Detection of Uneven Alveolar Ventilation During a Single Breath of Oxygen; a New Test of Pulmonary Disease, Am. J. Med. **10**: 408, 1951.
2. Darling, R. C., Cournand, A., and Richards, D. W., Jr.: Studies on the Intrapulmonary Mixture of Gases: III. Open Circuit Method for Measuring Residual Air, J. Clin. Invest. **19**: 609, 1940.
3. Wright, W. W., and Michelson, E.: in Comroe, J. H., Jr., editor: Bronchospirometry, Methods in Medical Research, Chicago, 1950, The Year Book Publishers, Inc., vol. 2, p. 82.
4. Alexander, R. S., and Reydman, M. M.: A Single Test of Pulmonary Function Employing the Oximeter, J. THORACIC SURG. **25**: 95, 1953.

THE POSTOPERATIVE STATUS OF THE DEPENDENT LUNG

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FREDERICK W. DASCH, M.D.
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A ROENTGENOGRAPHIC study of the lungs in the period immediately after thoracic surgery was undertaken because of interest aroused by differences in re-expansion noted in the lung upon which surgery had been performed. Unexpected findings in the contralateral lung in the first few cases studied led to further roentgenographic investigation of the effects of the surgical position per se on the aeration of the dependent lung.

A review of the literature failed to reveal any similar studies although several studies [1-5] were reported on surgical position in relation to respiratory acidosis, carbon dioxide retention, etc.

METHOD OF STUDY

The study was conducted at Eagleville Sanatorium, Eagleville, Pennsylvania, a 200-bed sanatorium devoted to the treatment of pulmonary tuberculosis. The proximity of the Radiology Department to the operating rooms made it possible to expose roentgenograms on the unconscious patient within 3 minutes of wound closure. Patients were taken in their respective operative positions on the operating table directly to the Radiology Department across the hall from the surgical suite.

Thirty-three tuberculous patients were studied, 19 of whom were males. Resectional surgery had been performed in 32 cases. The lateral decubitus position had been used in 27 instances and the prone or supine position in 5 cases. The list of surgical procedures follows:

Pneumonectomy	5
Lobectomy	9
Segmental resection	11
Wedge resection	7
Closed pneumonolysis	1
	33

The first 16 patients, all of whom had been positioned for surgery in the lateral decubitus, were turned to the supine position a moment before film exposure. Anteroposterior exposures were made at the peak of inspiration. Endotracheal tubes had been disconnected from the anesthesia apparatus but remained in situ to maintain patent airways.

The abnormalities noted in these films aroused curiosity as to whether or not still greater changes would be found if roentgenograms were taken without turning the patients.

Therefore, in the subsequent 11 cases, films were taken in the lateral decubitus position in which surgery had been performed. Controlled manual respiration was maintained by the anesthesiologist with the endotracheal tube

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still connected to the anesthesia apparatus. After films were taken in the lateral decubitus position the patients were turned to the supine position and other roentgenograms were exposed.

Of the 5 pneumonectomies, 4 had been performed in the prone (Overholt) position, and 1 in the supine position. All 5 were x-rayed in their respective positions while on controlled respiration. The patient whose operative procedure had been done in the supine position was turned to the lateral decubitus position and a film was exposed immediately. Because of the nature of the Overholt position, it was not possible to shift the position of the 4 patients operated on in the prone position.

Three different anesthesiologists participated in this study. Pentothal sodium was used for induction. Syncurine was added before intubation. Closed circuit manually controlled ether-oxygen was administered during the resection. Nitrous oxide-oxygen was substituted during closure. Secretions were aspirated and the lungs were reinflated at frequent intervals throughout the operation.

RESULTS

All roentgenograms on patients whose surgical procedures had been carried out in the lateral decubitus position revealed evidence of increased densities which varied from patchy infiltrations (Fig. 1, *B*, Case 1), to complete homogeneous opacity of the dependent lung. The exposed lung was consistently free of such abnormalities (Fig. 2, *B*, Case 2).

These findings were more pronounced in the films taken when the patients had not been turned from the lateral decubitus position (Fig. 2, *B*, Case 2). There was marked clearing immediately after turning to the supine position (Fig. 2, *C*, Case 2).

In patients operated upon in the supine or prone position the contralateral lung was well aerated and free of compression. However, in Case 3, poor aeration in the remaining lung developed immediately when the patient was turned to the right lateral decubitus position for 1 minute (Fig. 3, *C*).

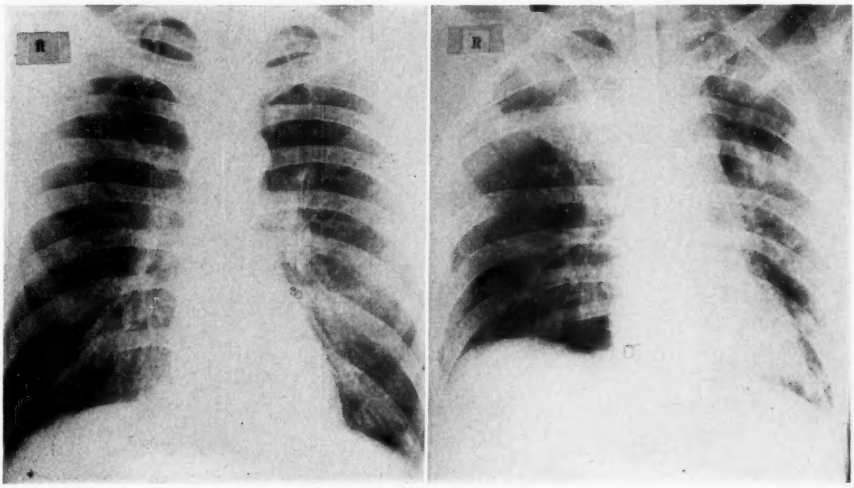
In the patient who had had closed pneumonolysis, roentgenograms of the contralateral lung revealed increased density throughout the lung (Fig. 4, *B*, Case 4).

DISCUSSION

The roentgenographic abnormalities of the dependent lung in patients operated upon in the lateral decubitus position were probably due largely to three factors—compression from above downward by the weight of the mediastinal structures, elevation of the diaphragm by weight of abdominal contents (Fig. 4, *B*, Case 4), and immobilization of the dependent hemithorax by the weight of the patient's body.

The vector effect of the three factors mentioned greatly reduces the volume of the dependent lung and, therefore, alveolar ventilation. Restricted movement favoring accumulation of secretions and patchy atelectasis may also be involved. In our small series the effects described were independent of the extent of tuberculous involvement, the type of surgery, or the duration of anesthesia.

It is of interest to note the rapidity with which the generalized haziness developed in the dependent lung in Case 3 when the patient was turned from



A. *B.*

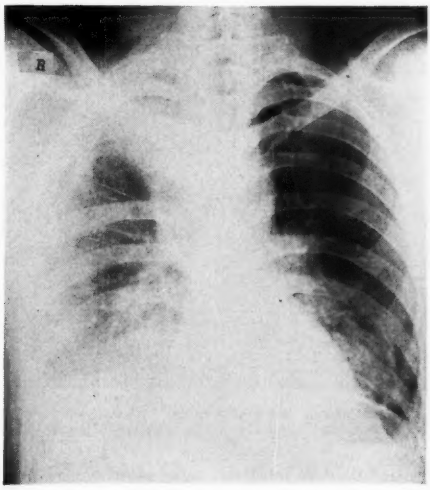


Fig. 1.—Case 1, J. J., a 32-year-old white man.

A, Preoperative film June 14, 1953, reveals a thin-walled cavity in the right apex, sputum positive.

B, Anteroposterior roentgenogram exposed with patient in the supine position immediately following resection of the apical and posterior segments of the right upper lobe, June 16, 1953. Operation had been performed in the left lateral decubitus. Note areas of patchy density in the left lung which had been dependent during surgery.

C, Erect film taken on the first postoperative day, June 17, 1953, with the left lung clear and normal postoperative reaction in the right lung.

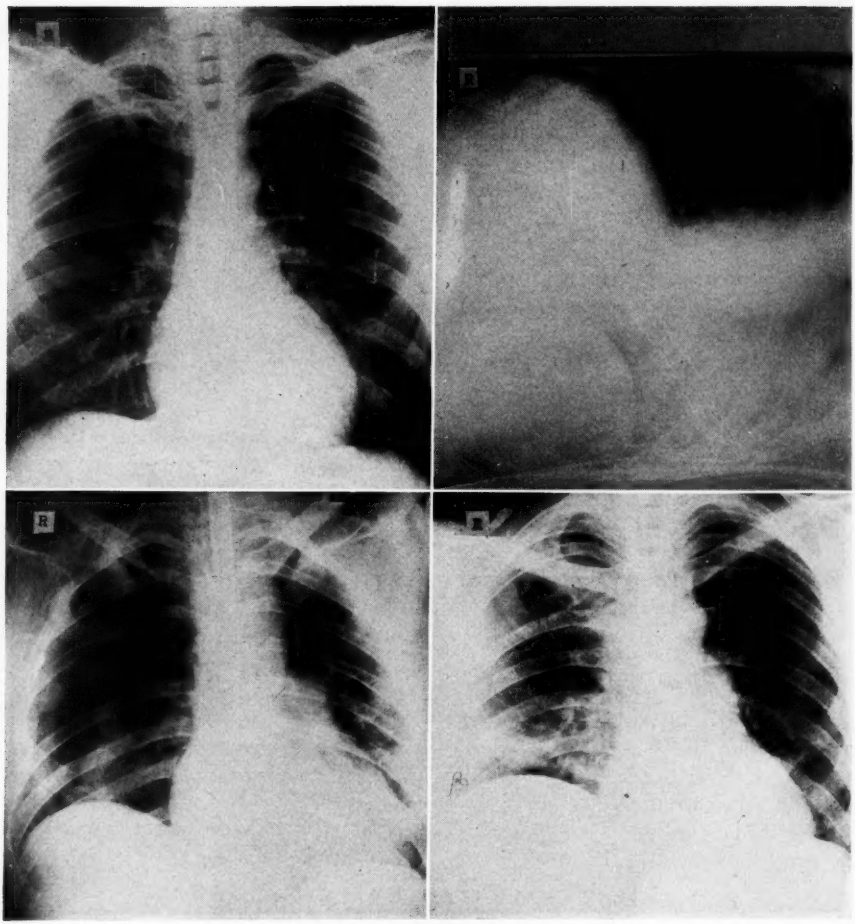


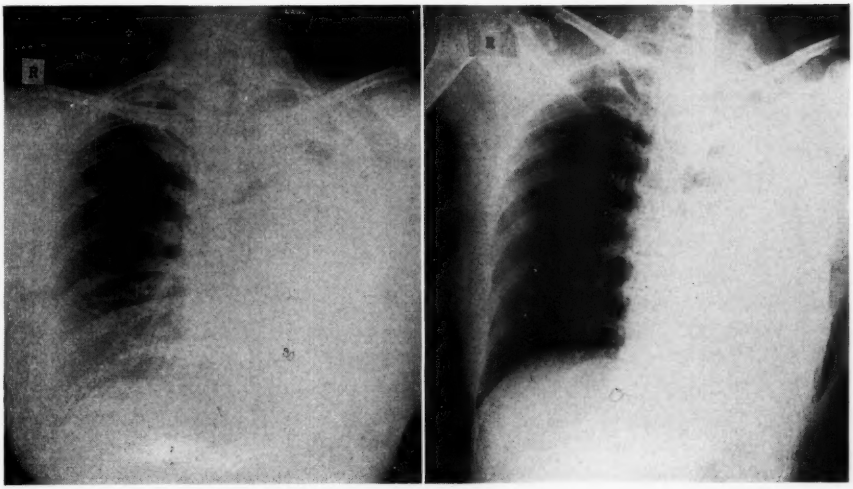
Fig. 2.—Case 2, A. S., a 35-year-old white man.

A, Preoperative film taken on April 24, 1954, reveals disease in the right apex, sputum positive.

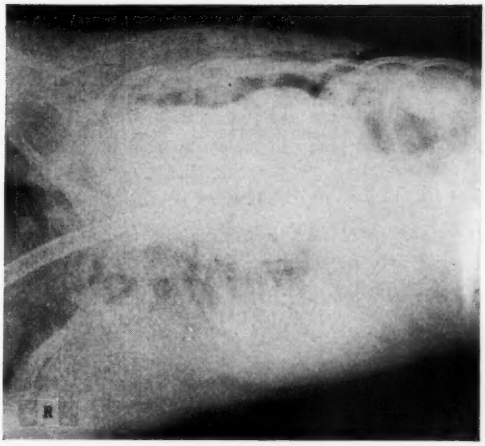
B, Left lateral decubitus film exposed immediately following resection of the apical and posterior segments of the right upper lobe, April 26, 1954. Operation had been performed in the left lateral decubitus.

C, Anteroposterior film exposed in the supine position within 1 minute after the patient was turned from the left lateral decubitus position. Note that clearing has started in the left lung.

D, Erect posteroanterior film taken the first postoperative day reveals clear left lung and normal postoperative reaction in the right lung.



A. *B.*



C.

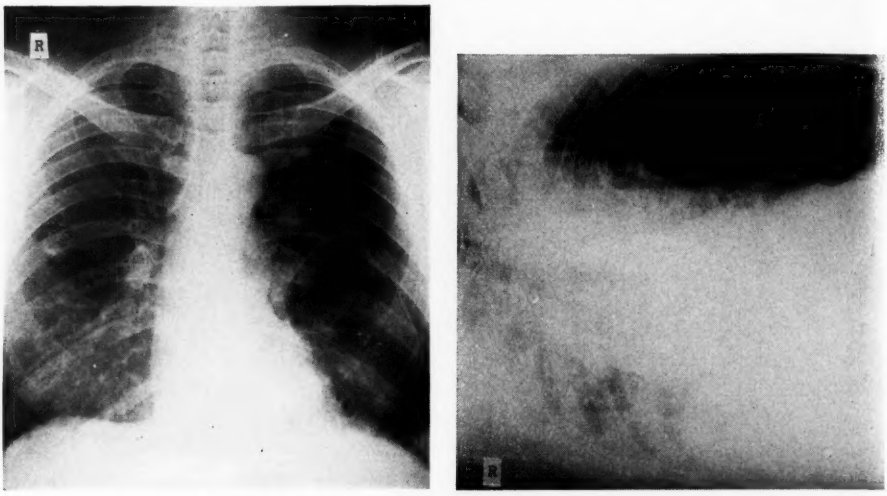
Fig. 3.—Case 3, J. G., a 57-year-old white woman.

A, Preoperative film taken on April 17, 1954, reveals a completely destroyed and atelectatic left lung.

B, Anteroposterior roentgenogram exposed with patient in the supine position immediately following left pneumonectomy on April 19, 1954. Operation had been performed in the supine position. Note the well-aerated contralateral right lung. Density of the left thorax caused by saline placed in the left pleural space before closure.

C, Right lateral decubitus film taken within 1 minute after the patient had been turned from the supine position. Note the rapidity with which changes in the right lung have occurred.

the supine to the lateral decubitus position. The rapidity with which the dependent lung reverted to normal when the patient was turned from the lateral decubitus to the supine position was also impressive. (Fig. 2, *C*, Case 2.)



A. *B.*

Fig. 4.—Case 4, G. C., a 47-year-old Negro man.

A, Preoperative film exposed on April 3, 1954, reveals a left artificial pneumothorax with apical adhesions.

B, Anteroposterior film exposed in the right lateral decubitus position immediately after left thoracoscopy under Pentothal sodium anesthesia. The adhesions could not be cut. Note the changes in the dependent right lung and the elevation of the right diaphragm.

SUMMARY

1. Thirty-three patients who had undergone thoracic surgery were x-rayed immediately postoperatively in various positions.

2. The changes in the dependent lung are described.

3. Possible mechanisms involved in changes observed are described.

We wish to thank Drs. Seymore Schotz, Demetrius Kostrubiak, and Basil Rozhin, the participating anesthesiologists, for their assistance, and Dr. J. Gershon-Cohen and his associates for their roentgenographic interpretation. Also our thanks are expressed to Dr. Katharine R. Boucot, for her editorial assistance.

REFERENCES

1. Etsten, Benjamin: Respiratory Acidosis During Intrathoracic Surgery: The (Overholt) Prone Position, J. THORACIC SURG. **25**: 286-299, 1953.
2. Frank, Howard A.: Circulatory and Respiratory Effects of Continuous Versus Phasic Lung Inflation in Open-Chest Dogs. Description of Apparatus for Application of Positive Pressure, J. THORACIC SURG. **23**: 465-478, 1952.
3. Taylor, F. H., and Roos, A.: Disturbance of Acid-Base Balance During Ether Anesthesia With Special Reference to Changes Occurring During Thoracic Surgery, J. THORACIC SURG. **20**: 289-295, 1950.
4. Gibbon, J. H., Jr., Allbritten, F. F., Jr., Stayman, J. W., Jr., and Judd, J. M.: A Clinical Study of Respiratory Exchange During Prolonged Operations With an Open Thorax, Ann. Surg. **132**: 611-625, 1950.
5. Beecher, H. K., and Murphy, A. J.: Acidosis During Thoracic Surgery, J. THORACIC SURG. **19**: 50-70, 1950.

PNEUMONECTOMY IN PULMONARY TUBERCULOSIS WITHOUT THORACOPLASTY

Clinical Results and Lung Function Studies

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WHEN we began treating pulmonary tuberculosis with resection, we had to decide whether or not supplementary collapse was needed. From a study of the literature it appeared that the majority of authors, following the ideas of Overholt and associates.[1, 2] and Sweet,[3] were of the opinion that this was indicated, either by means of phrenic exeresis or by thoracoplasty. Overholt spoke of "tailoring the chest to fit the lung." By omitting supplementary collapse the patients would be exposed to certain risks:

1. The chance of developing tuberculous empyema would increase.
2. Bronchial fistula might arise by traction of the bronchial stump.
3. By distention of the remaining lung tissue, the tuberculous foci therein could reactivate.
4. The remaining distended lung tissue would be sensitive to a new generalized tuberculosis infection.
5. By overdistention, the remaining lung tissue could develop emphysema.

Although these risks were real, it appeared to us that it was a most mechanical conception to say that all these complications could be prevented by phrenic exeresis or thoracoplasty. Other authors, too, (Sarot,[4] Sellors and Hickey,[5] Jones and Robinson[6]) were skeptical regarding the routine use of supplementary collapse, although they did not state precisely its exact indications. We decided, therefore, not to use supplementary collapse and study our results. This point of view has also been adopted by other Dutch authors (Eerland,[7] and Nuboer[8]).

Between October, 1949, and October, 1953, 600 lung resections were done at the Berg en Bosch Sanatorium for the treatment of pulmonary tuberculosis.* By Oct. 1, 1954, the mortality rate was 3.1 per cent—11 patients died as the result of the operation, 4 as the result of complications, and 4 because of a relapse after discharge from treatment (Hirdes and Stegerhoek[9]). Included in these 600 resections were 83 pneumonectomies and 23 pleuropneumonectomies (106, or 17.7 per cent). Of these, 45 were performed on men and 61 on women. Their ages are given in Table I.

From the Sanatorium "Berg en Bosch."
Received for publication Feb. 23, 1955.

*Surgeon: M. C. A. Klinkenbergh, M.D.; co-workers: A. G. Brom, M.D., and A. M. L. E. S. Schaepkens v. Riempst; bronchoscopist: C. R. J. Versteegh, M.D.; and anesthetist: H. de Zwaan.

TABLE I

	0-10 YEAR	10-20 YEAR	20-30 YEAR	30-40 YEAR	40-50 YEAR	50-60 YEAR	TOTAL
Number	2	10	40	30	16	8	106
Died	1	–	2	1	1	1	6
			>1*				>1*
Relapse	–	–	2	–	–	–	2

*This case must be added to both groups.

During sanatorium treatment 6 patients died (6 per cent). After discharge 3 patients (3 per cent) had a relapse, one of whom died.

The indications for operation are shown in Table II.

TABLE II

	NUMBER	DURING SANATORIUM TREATMENT		AFTER DISCHARGE FROM TREATMENT	
		DIED	EMPYEMA	RELAPSE	DIED
Primary tuberculosis	3	1	–	–	–
Ulcerative phthisis	8	–	–	–	–
Tuberculomas	1	–	–	–	–
Tuberculous bronchitis	13	1	–	1	–
Cavities	49	–	1* 1	1	1
Destroyed lung and/or empyema	32	2	1* 2	–	–
Total	106	4	2* 3	2	1

*This number must be added to both groups.

DIED

Primary tuberculosis:

1.—H. J., a boy, 4 years old. There was a gland perforation in the right main bronchus with obstruction pneumonia in the entire right lung. He died from shock on the second day following operation.

Tuberculous bronchitis:

2.—L. M., a woman, 40 years old. There was stenosis of the left main bronchus. She died on the twelfth day following operation from bronchopneumonia of the right lung.

Cavities:

3.—B. R., a man, 54 years old. Pneumonectomy was performed on the left side. During the operation, a large cavity perforated. A tuberculous empyema developed with a bronchial fistula. He died eight months after operation.

4.—J. L., a woman, 29 years old. The patient had pneumonectomy performed on the left side and had had previous dissemination in the right lung. She was discharged in good condition. A year after the operation, she developed a cavity on right side. The patient died two years later.

Destroyed lung:

5.—B. B., a man, 28 years old. The patient had a destroyed lung with tuberculous empyema and bronchial fistula on the right side. After pleuropneumonectomy, tuberculous empyema appeared again plus a bronchial fistula. He died eighteen months after operation.

6.—P. R., a woman, 38 years old. A right pneumonectomy was performed. The patient died on the fifteenth day following operation as the result of heart and lung insufficiency.

7.—V. W., a man, 29 years old. The patient had right pneumonectomy performed. He died on the tenth day following operation as the result of anuria (amyloidosis of the kidneys).

EMPYEMAS

Cavities:

I.—M. H., a woman, 37 years old. A right pneumonectomy was performed. On the third day after the operation staphylococcic empyema developed. The patient was cured after intrathoracic treatment with Chloromycetin.

II.—B. R., a man, 54 years old. No. 3 of deceased patients.

Destroyed lung:

III.—B. B., a man, 28 years old. No. 5 of deceased patients.

IV.—P. S., a man, 49 years old. Pleuropneumonectomy was performed on the right side. On the fifth day following operation he developed staphylococcic empyema. The patient was cured after intrathoracic treatment with Chloromycetin.

V.—L. S., a man, 54 years old. The patient had a right pleuropneumonectomy performed. Staphylococcic empyema developed on the sixth day after operation. He was cured after thoracoplasty with flap drainage.

We believe there is no connection between the cause of death of the 7 deceased patients and the fact that we did not do a thoracoplasty after the pneumonectomy, with the exception, perhaps, of Case 4.

Altogether, we had only 5 cases of empyema, of which 3 resulted from staphylococci and 2 from tubercle bacilli. Only the last 2 patients developed bronchial fistula. Tuberculous empyema with bronchial fistula developed in only 2 per cent of our patients on whom pneumonectomy or pleuropneumonectomy was performed. These complications developed so soon after operation that it is not apparent to us how we could have prevented them by doing a thoracoplasty. The technique of closing the bronchial stump and the sensitivity of the tubercle bacilli to antibiotics are of much greater significance than the thoracoplasty.

The technique of our surgeon, Dr. Klinkenbergh, in closing the bronchial stump is simple. Two continuous sutures, which cross each other in the same manner as the laces of a shoe, close off the bronchus. If edema of the mucous membrane develops or if the patient coughs, these threads constrict still more, preventing leakage. The vascularization of the bronchial stump is not interfered with by these sutures, so within a short time the margins of the mucous membrane grow together. The bronchial stump is also covered with pleura and embedded in the mediastinum (Figs. 1, 2, and 3).

If the patient can be effectively protected with antibiotics, the chance of the development of a tuberculous empyema is small. Not one of our patients, prior to the operation, was resistant to streptomycin. We stress this point because the figures demonstrate how important this fact is in preventing tuberculous empyema. The thoracic cavity is not drained after the operation. During

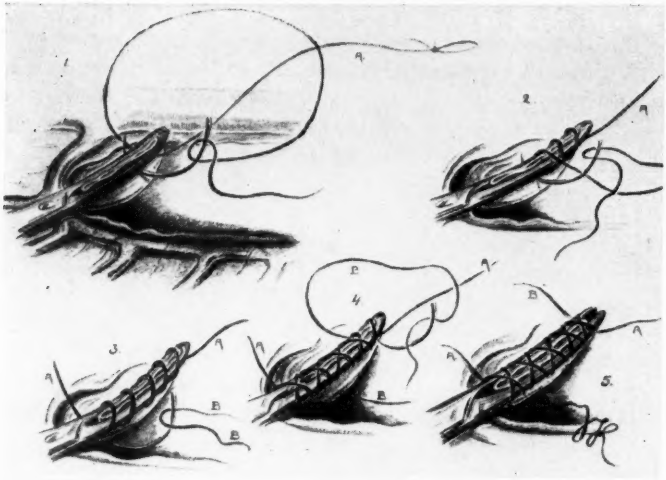

Fig. 1.

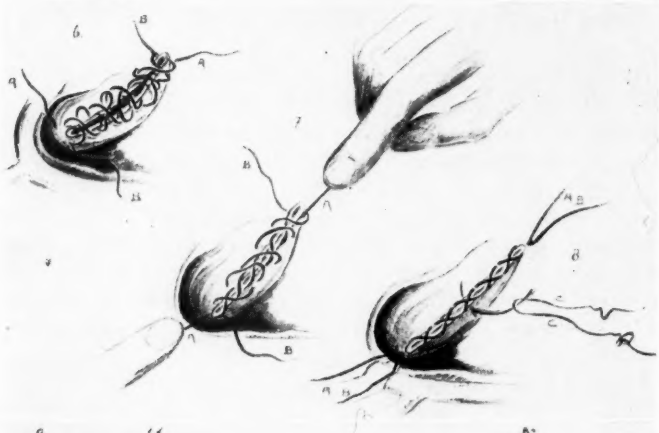

Fig. 2.

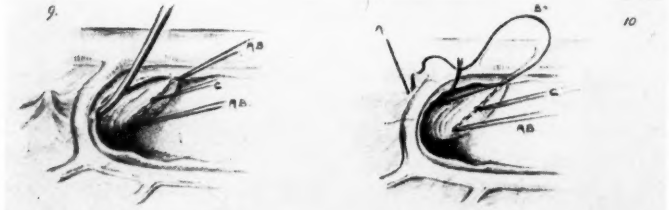

Fig. 3.

a period of some weeks following surgery, antibiotics (streptomycin para-aminosalicylic acid, and isonicotinic acid hydrazide) are not only administered by mouth or intramuscularly, but are also injected every day, intrathoracically.

The results at the time of discharge of these 106 patients are shown in Table III.

TABLE III

	NUMBER	VERY GOOD	GOOD	UNSATISFACTORY	BAD	DIED
Primary tuberculosis	3	2	–	–	–	1
Ulcerative phthisis	8	7	1	–	–	–
Tuberculomas	1	1	–	–	–	–
Tuberculous bronchitis	13	10	2	–	–	1
Cavities	49	45	2	–	1	1
Destroyed lung and/or empyema	32	26	2	1	–	3
TOTAL	106	91	7	1	1	6
PER CENT	100	86	6	1	1	6

Ninety-one patients recovered completely. Their postoperative sputum cultures were repeatedly negative. Only 7 patients still had a positive sputum culture. This was caused mostly by small foci in the remaining lung, which resulted from bronchogenic spread. Considering the serious clinical picture and the high bacterial count of the sputum of these 7 patients prior to surgery, we classify their results as "good." In one patient the result was unsatisfactory (Case V of empyemas). His lung function was considerably reduced after we had to do a ten-rib thoracoplasty with flap drainage in order to control his staphylococcic empyema. In another patient the result was bad. He had a small thin-walled cavity in the remaining lung which increased immediately after operation. In the latter patient treatment is being continued. Six patients have died. The follow-up on the 99 patients is given in Table IV.

TABLE IV

DURATION OF THE FOLLOW-UP	NUMBER OF PATIENTS	RELAPSE
<1 year	99	1*
>1 year	94	1
>2 years	63	1
>3 years	33	0
>4 years	17	0
>5 years	5	0
>6 years	2	0

*Died.

In the follow-up, reactivation in the remaining lung must be considered. Immediately following pneumonectomy we observed two cases of bronchogenic spreads. In both patients these spreads resorbed in a short time under antibiotic therapy. After discharge three reactivations occurred, each with the formation of cavities. One patient died (Case 4 of the mortality list); 2 are again under treatment and have a dubious prognosis.

Considering the extent of disease in the patients operated upon and the poor resistance many of them had to tuberculosis, the reactivations were few and need not be attributed to stretching of the remaining lung.

TABLE V

NUMBER OF SUBJECT (FIGS. 4-14)	SUBJECT	SEX	AGE (YEARS)	STATURE (M.)	PNEUMONECTOMY R/L	THORACOPLASTY	POSITION OF DIAPHRAGM	DISPLACEMENT OF MEDIASTINUM† AND TIME BETWEEN PNEUMONECTOMY AND LUNG FUNCTION TESTS IN MONTHS									
1*	T. M.	m	38	1.79	L	–	Phrenic exeresis during pneumonectomy	I	5	I	69	I	102				
2	R. A.	f	28	1.67	L	–	Phrenic exeresis during pneumonectomy	0	16	0	28	0	57	0	75	0	86
5	S.-S.	f	32	1.53	L	–	–	I	8	I	48	I	76				
6	J. V.	f	26	1.83	L	–	–	III	16	III	42						
7	M. E.	f	19	1.74	R	2d-7th rib included during pneumonectomy	–	0	23	0	39	0	51				
8*	A. S.	f	23	1.57	L	–	–	I	7	I	18	I	30	I	49		
9*	S. H.	f	24	1.53	L	–	High position of left diaphragm	I	7	I	16	I	29				
10	C. D.	m	59	1.73	R	–	Phrenic exeresis during pneumonectomy	0	16	0	29	0	49	0	60		
12	A. H.	f	24	1.59	R	–	–	II	8	II	32						
13*	W. S.	m	47	1.76	R	–	–	0	7	0	22	0	35	0	53		
16*	G. G.	f	20	1.42	L	–	–	I	7	I	21	I	37	I	52		
17*	W. B.	m	23	1.76	L	–	High position of left diaphragm	0	6	0	12	0	29	0	42	0	52
18	J. A.	m	24	1.78	R	–	–	0	6	0	23	0	51				
19*	W.-N.	f	39	1.65	R	–	–	0	5	0	18	0	36				
21*	K.-M.	f	47	1.55	L	–	–	I	4	II	17	II	35				
22*	B. L.	f	17	1.77	L	–	–	0	4	I	17	I	35	I	47		
23	H. H.	m	34	1.78	R	–	–	0	33								
26*	J. V.	m	32	1.65	L	1st-7th rib 10 months before pneumonectomy	High position of left diaphragm	0	10	0	32	0	46				

27*	H. M.	m	23	1.68	L	–	High position of left diaphragm	I	8	I	25	I	44			
28*	J. G.	m	41	1.80	L	–	–	0	7	0	18	0	35	0	45	
30*	J. K.	m	20	1.75	L	–	–	0	9	0	24	0	37			
31*	D. H.	f	34	1.57	L	–	–	0	4	0	23	0	36	0	45	
33	T. B.	m	33	1.73	L	–	–	0	9	0	41					
34*	J. L.	m	47	1.84	L	1st-7th rib 2 years before pneumonectomy	–	0	6	0	18	0	31			
35	M. D.	f	29	1.67	R	–	–	I	19	I	39					
37*	R. B.	f	24	1.72	L	–	–	I	6	II	22	II	37			
40*	D. G.	f	42	1.75	L	–	Phrenic exeresis 21 years before pneumonectomy	0	6	0	22	0	37			
41	C. K.	m	29	1.81	R	–	–	III	9	III	24					
42*	J. V.	m	31	1.72	R	–	–	I	8	I	25	I	35			
43*	J. C.	f	26	1.72	R	–	–	0	6	I	19	I	34			
46*	R. K.	f	20	1.57	R	–	–	III	4	III	24	III	36			
47*	J. B.	f	37	1.64	L	–	–	0	5	0	17	0	33			
48*	W. B.	m	53	1.78	R	–	–	0	10	0	23	0	34			
55	J. H.	m	36	1.87	L	–	–	II	7	II	21					
57	J. A.	f	22	1.62	L	–	–	II	3	III	19					
58	J. D.	m	31	1.66	L	–	High position of left diaphragm	0	7	0	20					
73	R.-K.	f	46	1.65	L	–	–	0	5	0	24					
74	J. L.	m	27	1.76	R	–	High position of right diaphragm	0	10	0	24					

*The results of the lung function tests of these patients are summarized in Fig. 14.

† 0=hardly any displacement.
I=a slight displacement.
II=a considerable displacement.
III=very pronounced displacement.

The question remains, however, as to whether stretching of the remaining lung gives rise to emphysema and reduces lung function. Some authors express the view that this is, indeed, the case. They base this opinion on the studies of Longacre and co-workers,[10-12] Bremer,[13] and Cournand and associates.[14-16] It appears, however, that these workers exercise great caution in their conclusions in this regard. Birath and associates[17] state, "The fears of emphysema that have been expressed in connection with this distention received only slight confirmation in the cases examined." Gaensler and Strieder[18] have also compared results of lung function in patients with and without thoracoplasty after pneumonectomy. They say, "Thoracoplasty accomplished just the opposite of the desired result. The range of the useful respiratory movement (VC) was greatly reduced, whereas the volume which merely dilutes the inspired tidal air (residual volume) was only slightly reduced. . . . [from 38 to 35 per cent of the total capacity.] Worst of all, the initial mean MBC of 58 liters per minute was permanently reduced to 46 liters per minute." Mathey and co-workers[19] emphasize this conclusion and consider the value of a thoracoplasty after pneumonectomy very doubtful.

We also studied the function of the remaining lung. In 38 of the 63 patients who underwent pneumonectomy more than two years ago (Table IV), we have been able to do, and to repeat at different times, lung function tests. These 38 patients are listed in Table V. Twenty patients were women, 18 men. Pneumonectomy was performed 24 times on the left side and 14 times on the right. Age at time of operation was as follows:

2 between 10 and 19 years
17 between 20 and 29 years
11 between 30 and 39 years
6 between 40 and 49 years
2 between 50 and 59 years

Three of these 38 patients had a thoracoplasty performed. In the female patient, No. 7, six ribs, the second up to and including the seventh, were removed during pneumonectomy. The operation was performed elsewhere and the patient was sent to our sanatorium because, notwithstanding the thoracoplasty, a small cavity developed contralaterally. In the male patients, Nos. 26 and 34, thoracoplasty, comprising seven ribs, was performed elsewhere ten months and two years respectively, prior to pneumonectomy. Both patients, however, had residual cavities in the collapsed area, so the diseased lung finally had to be removed.

In 4 patients phrenic exeresis was done. In patients No. 1 and No. 2, this was done during the pneumonectomy because we believed, at the beginning, it offered certain advantages—being influenced by the literature mentioned previously. These, however, were the only patients in whom phrenic exeresis was purposely done. In patient No. 10, the phrenic nerve was accidentally cut during surgery, and in patient No. 40, phrenic exeresis had been done twenty-one years previously. After examination, it appeared that patients Nos. 9, 17, 26, 27, 73, and 74 all had a highly positioned diaphragm on the side operated upon, probably as the result of contraction.

In the last column of Table V is detailed the displacement of the mediastinum from very slight to very pronounced. This column also shows how many months after pneumonectomy the follow-up examination of lung function took place. Only in 4 of the 38 patients (Nos. 6, 42, 46, and 57) was the displacement of the mediastinum very pronounced with development of a large mediastinal hernia. In 4 other patients (Nos. 12, 21, 37, and 55) the displacement was considerable, and in the remaining 30 patients only from slight to medium. All the patients with thoracoplasty, phrenic exeresis, or a high positioning of the diaphragm belonged in this latter group. The displacement of the mediastinum in most of the patients had already taken place at the initial examination a few months after the operation, and subsequently remained static, at least by x-ray.

For comparative study of the various data relating to the lung function tests, the normal vital capacity of all patients was calculated by the formulas of Anthony.[20]

Men: vital capacity = (caloric consumption per 24 hours) × (2.2 to 2.4)
Women: vital capacity = (caloric consumption per 24 hours) × (2.0 to 2.2)

For calculating vital capacity, the conversion factor of 2.4 was chosen for men and 2.1 for women. The caloric consumption per 24 hours is obtained from the formulas of Harris and Benedict with the ideal weight: w = s − 100:

Men: $h = 66.473 + 13.752 \times w + 5.003 \times s - 6.755 \times a$
Women: $h = 655.096 + 9.563 \times w + 1.850 \times s - 4.676 \times a$
h = caloric consumption per 24 hours in basal condition
w = weight in kilograms
s = height in centimeters
a = age in years

By substituting the formulas of Harris and Benedict in those of Anthony the following results are obtained:

Men: vital capacity in ml. $= 45.01 \times s - 16.21 \times a - 3141$
Women: vital capacity in ml. $= 23.97 \times s - 9.82 \times a - 632$

The thus calculated normal values approach closely the values obtained from the formulas of Baldwin, Cournand, and Richards.[21]

Men: vital capacity in ml. $= (27.63 - 0.112 \times a) \times s$
Women: vital capacity in ml. $= (21.78 - 0.101 \times a) \times s$

We have, however, given preference to the formulas of Anthony because they are more easily reproduced in a paper.

With the data from Table V and the previously indicated formulas, the normal vital capacity of each patient could be calculated. The normal total capacity, the residual volume, and the functional residual volume were obtained from Table VI, which was compiled with the aid of the data from Anthony,[20] Baldwin and associates,[21] Greifenstein and co-workers,[22] Gilson and Hugh-Jones,[23] and our own amendments.

The normal value for the right lung was fixed at 55 per cent of the calculated normal values for both lungs; for the left at 45 per cent. These have again been rated as the 100 per cent value of the remaining lung. The values obtained during the follow-up examinations have been converted on this basis.

TABLE VI. SUBDIVISION OF THE TOTAL CAPACITY (T.C.) IN ADULTS

AGE	VITAL CAPACITY* IN % T.C.	FUNCTIONAL RESIDUAL VOLUME IN % T.C.	RESIDUAL VOLUME IN % T.C.	RESERVE VOLUME IN % T.C.	TIDAL VOLUME IN % T.C.	COMPLEMENTARY CAPACITY IN % T.C.	USABLE PART OF VITAL CAPACITY* IN %
20-39 yrs.	75	40	25	15	11	49	83
40-49 yrs.	72	43	28	15	11	46	75
50-59 yrs.	68	47	32	15	11	42	70
60-70 yrs.	65	50	35	15	11	39	70

*Vital capacity=complementary capacity + tidal volume + reserve volume.

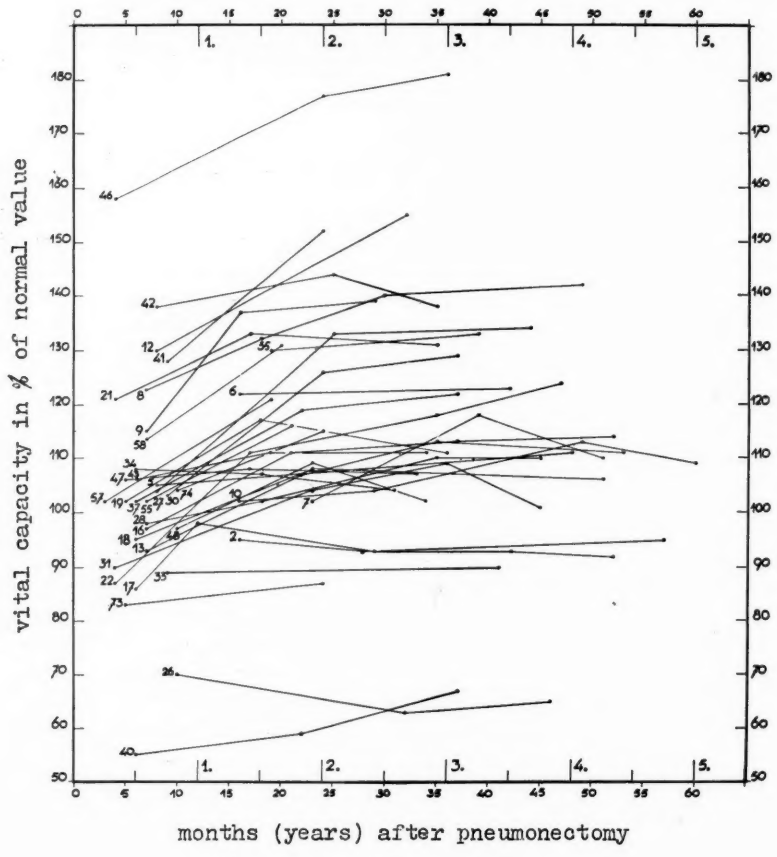

Fig. 4.

In accordance with this method Figs. 4 (vital capacity) and 5 (total capacity) have been compiled, whereby each line represents the volume alteration with the time after the operation. The numbers of the lines correspond to the numbers of the patients in Table V.

In Fig. 4 it is evident that the vital capacity of the majority of the patients operated upon exceeds 100 per cent and, with few exceptions, increases with the years. During the first year after operation (Fig. 6) the maximum vital capacity, expressed in percentages of the normal value, lies between 96 and 105 per cent; in the second year between 106 and 115 per cent. During the third and fourth years, because of smaller numbers and the large spread, a pronounced peak is lost.

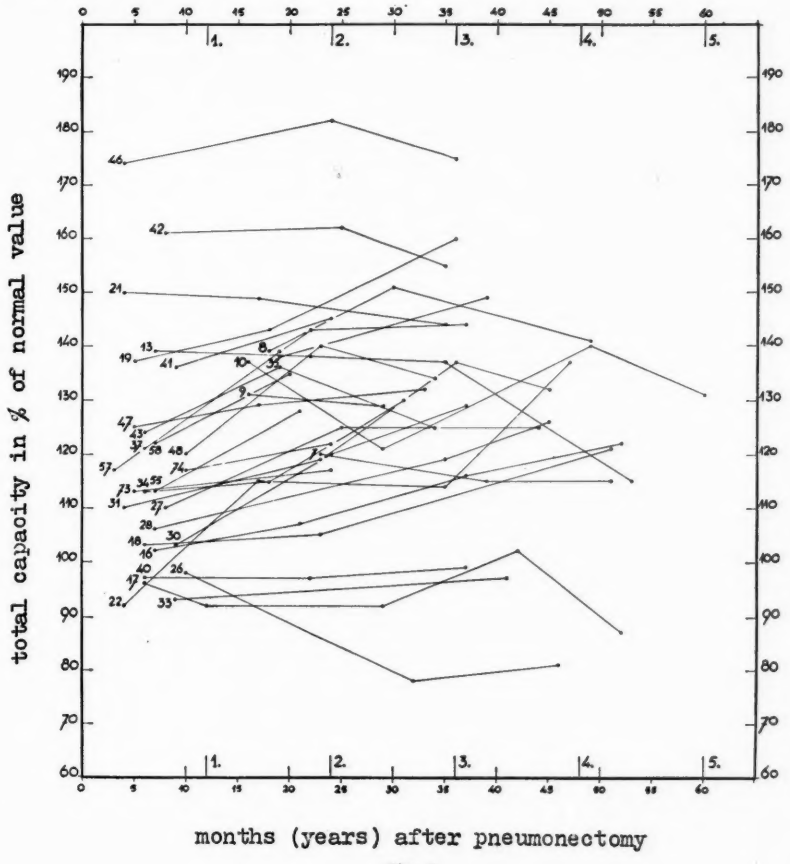

Fig. 5.

The lines of total capacity (Fig. 5) lie, for the greater part, considerably above 100 per cent. This means that the remaining lung, after pneumonectomy, when in the position of maximum inspiration is considerably larger than normal. Although a number of lines have an irregular course, there is, in the majority of the patients, a tendency to an increase of total capacity with time, though somewhat less pronounced than in the case of vital capacity. In the spreadings-curves of the total capacity in percentages of the normal value (Fig. 7), no distinct

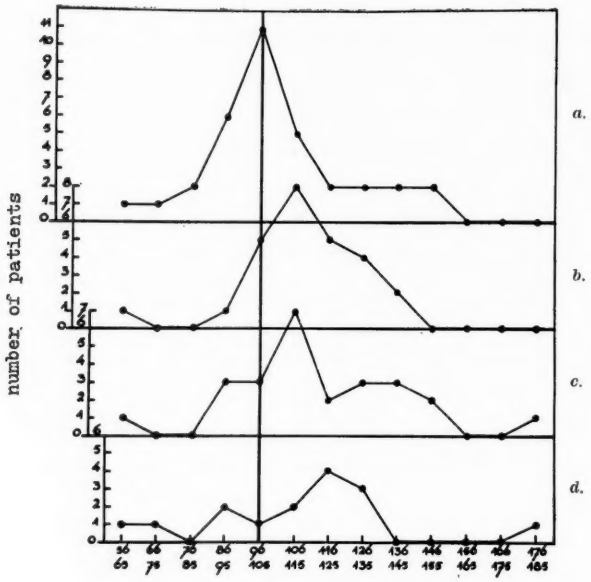

Fig. 6.

a, Within 12 months after operation: 32 patients
b, Between 13 and 24 months after operation: 26 patients
c, Between 25 and 36 months after operation: 25 patients
d, Between 37 and 48 months after operation: 15 patients

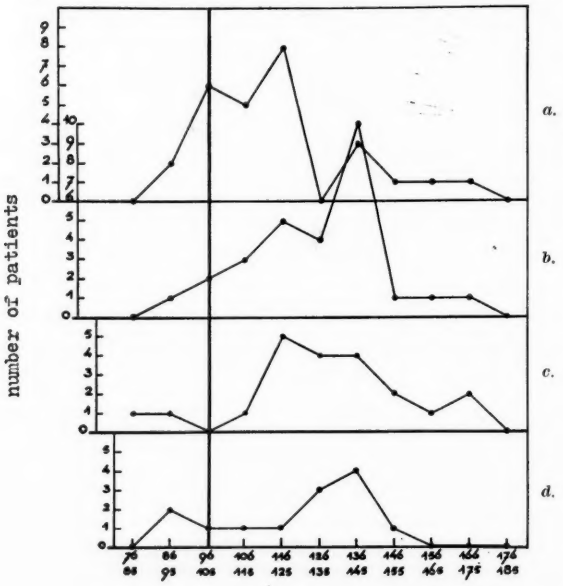

Fig. 7.

a, Within 12 months after operation: 27 patients
b, Between 13 and 24 months after operation: 28 patients
c, Between 25 and 36 months after operation: 21 patients
d, Between 37 and 48 months after operation: 14 patients

peaks are evident. What these curves do confirm, however, is that the values for total capacity lie much more above 100 per cent than those of vital capacity. This leads to the supposition that the residual volume after pneumonectomy must have increased.

The residual volume was determined by the method described by van Veen, Orie, and Hirdes,[24] and in accordance with the calculations of Bosch.[25] This method may be regarded as a modified oxygen-nitrogen method according to Christie,[26] whereby the gas exchange is measured with the aid of the diapherometer* of Noyons.[27] This method has the advantage of being reliable and it can be performed quickly without cumbersome analyses. The volumes are expressed in percentages of the total capacity. Although the residual volume of a number of patients (Fig. 8) exceeds the pathologic limit of 36 per cent, practically all the curves show a downward tendency. This observation is in opposition to the widely held opinion that, after pneumonectomy, emphysema develops.

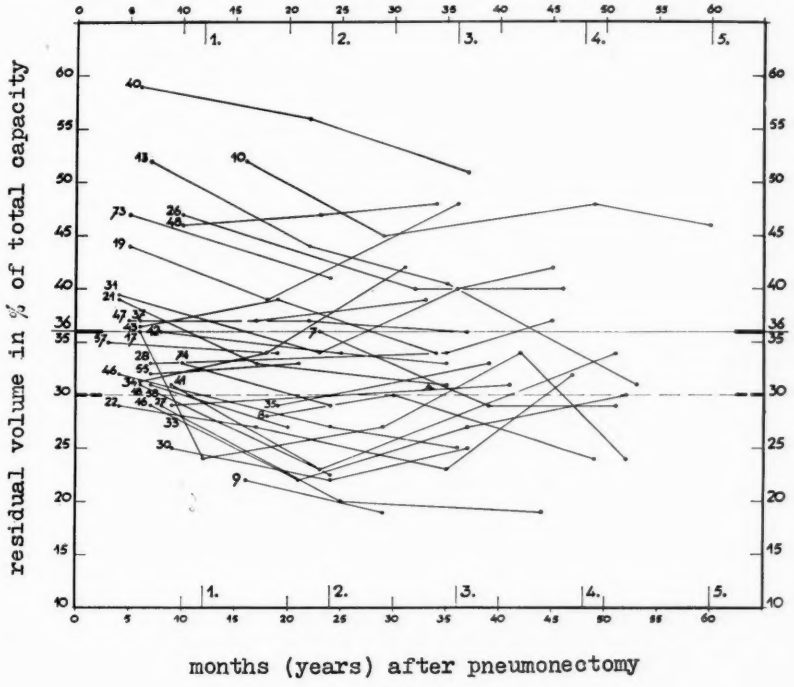

Fig. 8.

In the period of four years covered by our observations, this has not occurred. On the contrary, the relationship between vital capacity and residual volume, which can serve as a measure for the grade of emphysema, has become, in general, more favorable. This applies only, of course, to the patients who have been

*As manufactured and supplied by P. J. Kipp & Son, Delft, the Netherlands.

examined, of whom 30 were younger and 8 older than 40 years. The spreadings-curve (Fig. 9) confirms the fact that there is no increase of the residual volume in percentage of the total capacity.

The normal values of the maximum breathing capacity (M.B.C.) in liters were calculated according to the formula of Hirdes and van Veen:[28]

$$\frac{\text{vit. cap. in ml.}}{1{,}000} \times \text{usable part of the vit. cap. in \%} \times 30$$

The normal value for the usable part of the vital capacity employed in this formula is indicated in the last column of Table VI. This factor was introduced by Tiffeneau and Drutel[29] and is comparable to the "one second value" of Gaensler.[30] The values obtained for the M.B.C. were expressed in the same manner in percentages of the normal values of the remaining lung as is described

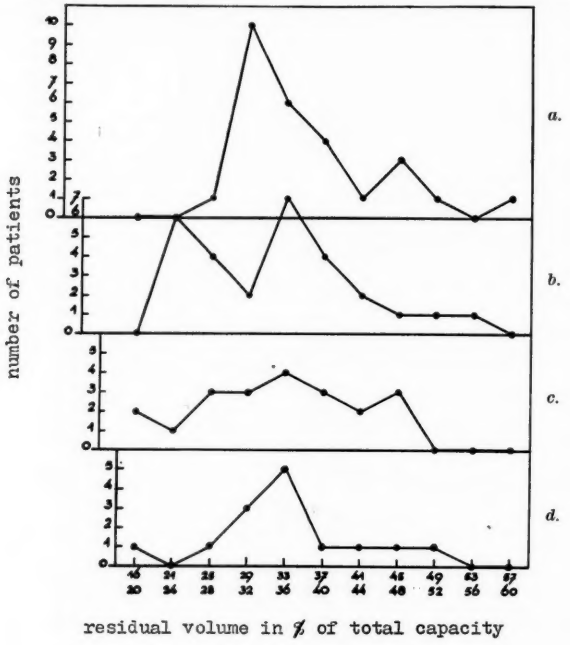

Fig. 9.

a, Within 12 months after operation: 27 patients
b, Between 13 and 24 months after operation: 28 patients
c, Between 25 and 36 months after operation: 21 patients
d, Between 37 and 48 months after operation: 14 patients

for both the vital capacity and the total capacity. In Fig. 10 the curves of the change of the M.B.C. of the individual patients have been reproduced. The majority lie around 100 per cent and here, once more, we observe, as in the case of the vital capacity, a tendency to increase in the course of time. In the spreadings-curve of the M.B.C. (Fig. 11) this is evident only in a reduction of the number of cases below 100 per cent. This shows that the ventilatory reserves become larger, a fact that is favorable for working conditions.

An important factor in answering the question as to whether, after pneumonectomy, emphysema does or does not develop is the elasticity of the lung. At present, however, there are no clinically usable methods by which this elasticity can be directly measured. The usable part of the vital capacity (Hirdes and van Veen,[28] and Tiffeneau and Drutel[29]), or the "one second value" of the vital capacity (Gaensler[30]), is, however, a factor for which normal values will

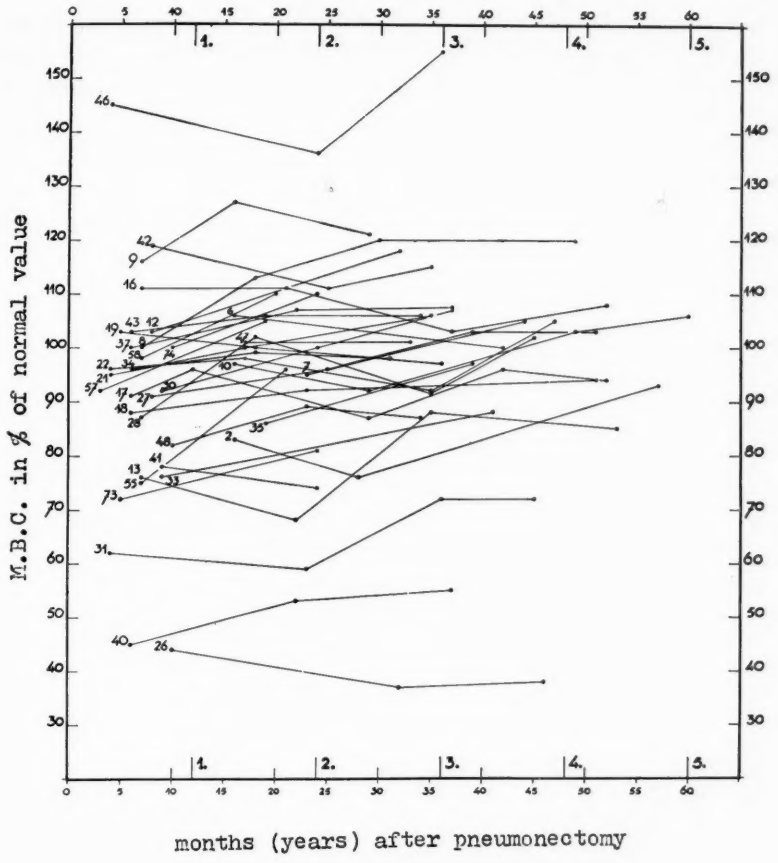

Fig. 10.

be secured only if the elasticity of the lung is not disturbed. In normal persons, the normal value lies between 70 and 90 per cent. With emphysema we see an increase in the residual volume in percentage of the total capacity and, simultaneously therewith, the usable part of the vital capacity (van Veen and Hirdes[31]) declines. In severely emphysematous lungs, restriction of the usable part of the vital capacity down to 30 per cent is no exception. In Fig. 12 is reproduced the course of the usable part of vital capacity in percentages for

each patient, including the time after pneumonectomy. Although there are a number of patients who have too low a value (<70 per cent), there is no general tendency to further reduction. This is confirmed by the spreadings-curve (Fig. 13). In general, therefore, one cannot speak of a loss of elasticity of the lung.

All of the patients marked with an asterisk in Table V had complete lung function tests in the first, second, and third years after operation, so their average values during successive years could be calculated. These average values of the total and vital capacity, the residual volume, the M.B.C., and the usable part of the vital capacity demonstrate clearly the tendency which has already been observed in the different curves (Fig. 14).

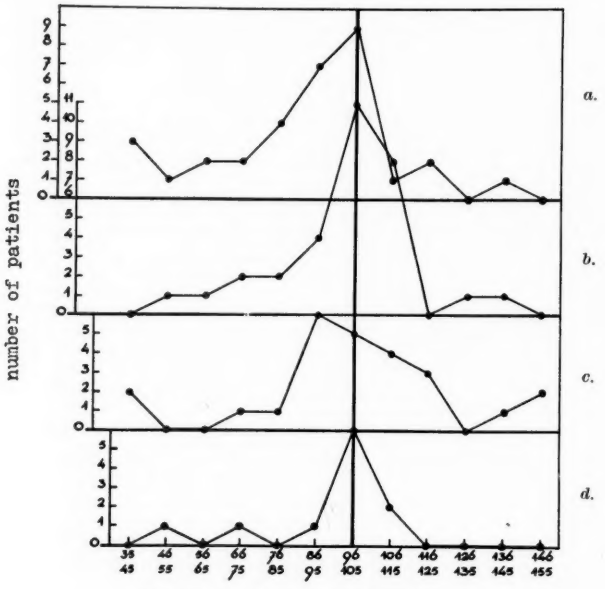

Fig. 11.

a, Within 12 months after operation: 30 patients
b, Between 13 and 24 months after operation: 31 patients
c, Between 25 and 36 months after operation: 22 patients
d, Between 37 and 48 months after operation: 11 patients

The fairly high percentage for the average residual volume of all 18 patients (line 1 of Fig. 14) requires further explanation. For this purpose we divided the patients into two groups (Table VII). The first group, of which the residual volume during the first examination was equal to or smaller than 36 per cent of the total capacity, was composed of 9 patients. The average age of this group was 25 years. The second group, with a residual volume larger than 36 per cent of the total capacity during the first examination, was also composed of 9 patients. Their average age was 39 years. The age of the pa-

tient at the time of pneumonectomy appears to influence directly the size of the residual volume after operation. There are no other distinct differences between the two groups which would explain the marked difference in the average residual volume. Each group includes 3 pneumonectomies on the right side and 6 on the left, and in each there is one patient in whom the pneumonectomy was complicated. In the first group, patient No. 34 had a seven-rib thoracoplasty two years prior to the pneumonectomy. This is the only patient whose residual volume in the ensuing years became larger. In the second group, in the female patient No. 40 phrenic exeresis was performed twenty-one years prior to and on the same side as the pneumonectomy. It is due to the phrenic exeresis that such an exceptionally large residual volume was seen in this patient.

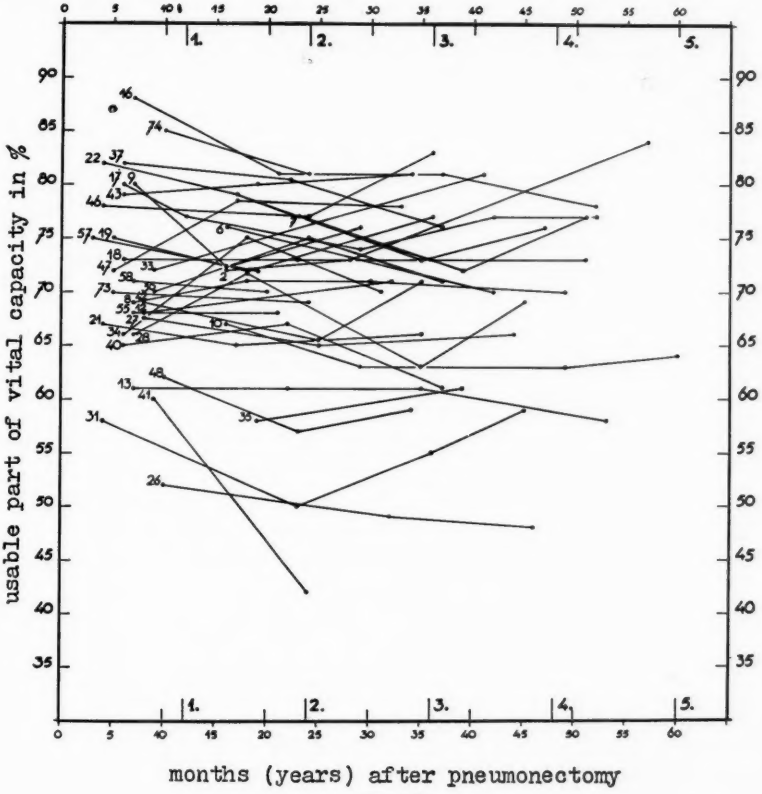

Fig. 12.

It is noted that for both groups the average residual volume diminishes with the years. This is observed not only in the younger patients with a small residual volume (Fig. 14, line 2), but also in the older patients with a large residual volume (Fig. 14, line 3). In the older patients, on the basis of the large residual volume, one might conclude that emphysema existed. As a rule,

TABLE VII. RESIDUAL VOLUME AT FIRST INVESTIGATION AFTER PNEUMONECTOMY

<36 PER CENT OF TOTAL CAPACITY					>36 PER CENT OF TOTAL CAPACITY				
		RESIDUAL VOLUME IN % TOTAL CAPACITY					RESIDUAL VOLUME IN % TOTAL CAPACITY		
SUBJECT	AGE (YEARS)	1ST YEAR	2ND YEAR	3RD YEAR	SUBJECT	AGE (YEARS)	1ST YEAR	2ND YEAR	3RD YEAR
16	20	29	22	27	13	47	52	44	41
17	23	36	24	27	19	39	44	39	48
22	17	29	27	23	21	47	39	33	31
27	23	29	20	19	26	32	47	40	40
30	20	25	22	25	31	34	39	34	40
34	47	31	34	42	37	24	37	37	36
42	31	36	34	33	40	42	59	56	51
43	26	36	37	31	47	37	37	37	39
46	20	32	27	25	48	53	46	47	48
Mean	25	31	28	28	Mean	39	44	41	41
		TOTAL CAPACITY IN % OF NORMAL VALUE							
Mean		119	128	129	Mean		122	126	129
		VITAL CAPACITY IN % OF NORMAL VALUE							
Mean		110	124	125	Mean		93	102	103
		USABLE PART OF VITAL CAPACITY IN %							
Mean		76	75	74	Mean		66	65	65
		M.B.C. IN % OF NORMAL VALUE							
Mean		105	106	109	Mean		78	79	84

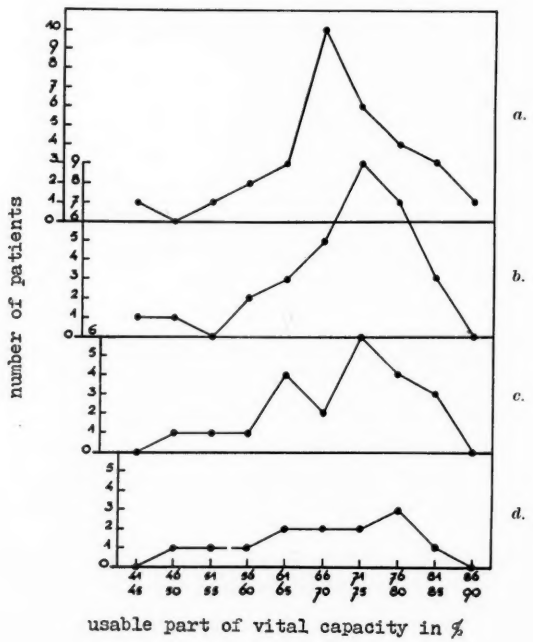

Fig. 13.

a, Within 12 months after operation: 31 patients
b, Between 13 and 24 months after operation: 31 patients
c, Between 25 and 36 months after operation: 22 patients
d, Between 37 and 48 months after operation: 13 patients

however, emphysema takes a progressive course and would, for that reason, be accompanied by an increase in residual volume through the years. This did not occur. In order to clarify this unexpected observation, the average total capacity and vital capacity in percentages of the normal value, and for each group separately, have been calculated, as well as the average usable part of the vital capacity and the average M.B.C. in percentages of the normal value (last lines of Table VII). It is evident that for both groups the total lung volume after pneumonectomy is enlarged to the same extent. In the youthful patients the usable part of the vital capacity is normal (>70 per cent); in the older patients it is low. The average M.B.C. in the older group lies, because of this,

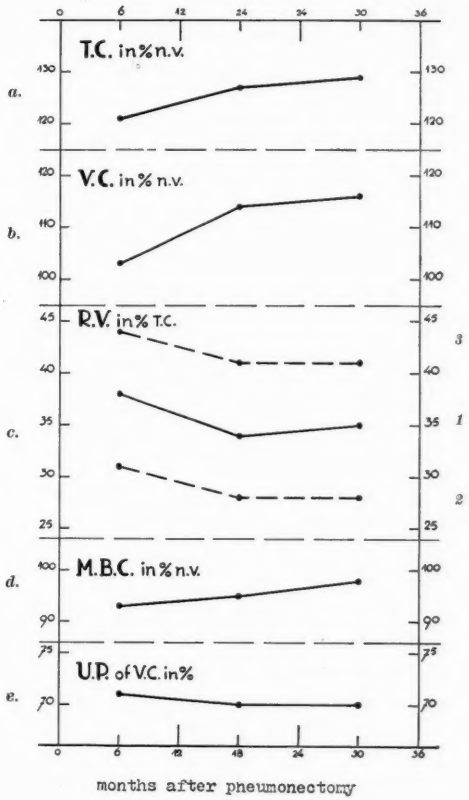

Fig. 14.

		MEAN VALUES FOR			NUMBER OF PATIENTS
		1ST YEAR	2ND YEAR	3RD YEAR	
a	total capacity in % of normal value	121	127	129	18
b	vital capacity in % of normal value	103	114	116	21
c	residual volume in % of total capacity	38	34	35	18
d	M.B.C. in % of normal value	93	95	98	20
e	usable part of vital capacity in %	71	70	70	20

considerably below the normal value. This resembles, up to a certain point, the so-called old age emphysema, for with normal and more elderly persons the total capacity remains constant and the residual volume increases at the cost of the vital capacity. Here the usable part of the vital capacity and the M.B.C. diminish and to an equal extent, precisely as in the group of older patients.

How can this be explained? It is possible that among the older group of patients there are those whose illness is of longer duration. The chances that disseminations in the remaining lung have taken place, which have given rise to fibrosis and emphysema with all the resulting functional changes, are hereby enhanced. We have not been able to find in the x-ray pictures any support for this assumption. Proof of an enlarged residual volume in the contralateral lung prior to pneumonectomy is absent, because no reliable method exists of measuring separately the residual volume and the total capacity of each lung.

Another possibility is that the larger residual volume in the older patients can be explained as due to a degenerative emphysema resulting from overstretching. Opposed to this is the fact that the large residual volume was observed at the first examination, within a year after pneumonectomy. Such a quick occurrence of these alterations in the structure of the lung tissue seems improbable. There are also the reduction in residual volume and the increase in vital capacity in the course of time—a recovery, therefore, in the direction of normal. These observations do not fit into the picture of a progressive degenerative emphysema.

It is quite probable that the mechanical disturbance of expiration, which results from every pneumonectomy, affords a better explanation of these facts. It is our belief that this disturbance in the case of young persons is compensated for in such a way that a normal residual volume can be attained, because the thoracic wall possesses possibilities of adapting itself as needed. Into this picture also fits the observation that, in the course of time, the residual volume declines when these patients learn to make further use of the adaptive possibilities. In older persons the disturbance of the breathing mechanism as the result of pneumonectomy will become apparent through an enlarged residual volume because the thoracic wall has become much less supple. It is quite understandable that here, too, some improvement can occur in the course of time.

If we grant such an important role to the functional unity of the thoracic wall and also to the diaphragm, it is not desirable to disturb this by thoracoplasty or phrenic exeresis. Moreover, this might result in the ventilatory function being considerably hindered, as is demonstrated in patients No. 26 and No. 40.

SUMMARY AND CONCLUSIONS

1. During sanatorium treatment, 6 of 106 patients died as the result of complications following pneumonectomy or pleuropneumonectomy (mortality 6 per cent).

2. Three cases of staphylococcic empyema and 2 of tuberculous empyema are reported. The first 3 patients recovered; the last 2, in whom bronchial fistulas developed, died.

3. Postoperative contralateral spread was observed in only 3 patients. In 2 this was transitory and the spreads disappeared within a short time under antibiotic therapy. In the third patient, there was an increase of an already existing cavity.

4. During a follow-up period of two to five years after discharge from treatment, 3 reactivations among 99 patients occurred, all 3 with cavity. One of these patients died.

5. In 38 patients, in whom lung function was checked during consecutive years postoperatively, the vital capacity and the total capacity increased. The M.B.C. also increased, although in lesser degree. The usable part of the vital capacity remained stationary. The residual volume was considerably higher in the group of older patients than in the group of younger patients. In both it diminished in the course of time. We believe that the results of the lung function tests in the older patients cannot be explained by fibrosis and emphysema of the remaining lung, as these findings existed before the pneumonectomy. Nor have any signs been found which point to a progressive emphysematous degeneration. It is probable that the mechanical disturbance of expiration explains these differences.

Two tuberculous empyemas and three reactivations among 106 patients do not justify thoracoplasty or phrenic exeresis as routine treatment after pneumonectomy for pulmonary tuberculosis. Nor do the results of the lung function studies justify these operations, although the function of older patients after pneumonectomy is slightly less favorable than that of younger patients. A reduction of the thoracic cavity by phrenic exeresis or thoracoplasty is undesirable as it disturbs the breathing mechanism and restricts ventilatory function. By omitting thoracoplasty we prevent the mutilating effect and psychic trauma of this operation on the patient.

REFERENCES

1. Overholt, R. H., and Wilson, N. J.: Pulmonary Resection in the Treatment of Pulmonary Tuberculosis, Am. Rev. Tuberc. **51**: 18, 1945.
2. Overholt, R. H., Langer, L., Szypulski, J. T., and Wilson, N. J.: Pulmonary Resection in the Treatment of Tuberculosis, J. THORACIC SURG. **15**: 384, 1946.
3. Sweet, R. H.: Lobectomy and Pneumonectomy in the Treatment of Pulmonary Tuberculosis, J. THORACIC SURG. **15**: 373, 1946.
4. Sarot, I. A.: Extrapleural Pneumonectomy and Pleurectomy and Pulmonary Tuberculosis, Thorax **4**: 173, 1949.
5. Sellors, T. H., and Hickey, M. D.: Excision of the Lung for Pulmonary Tuberculosis, Thorax **4**: 82, 1949.
6. Jones, J. C., and Robinson, J. L.: Pulmonary Resection in Tuberculosis: Its Hazards, Indications, and Results, J. THORACIC SURG. **20**: 882, 1950.
7. Eerland, L. D.: Resectiontherapy in Pulmonary Tuberculosis, Arch. chir. neerl. **5**: 213, 1953.
8. Nuboer, J. F.: Indications et Résultats du traitement chirurgical de la tuberculose pulmonaire par exérèse, à propos de 615 cas, Acta chir. belg. **5**: 439, 1953.
9. Hirdes, J. J., and Stegerhoek, C. I.: Resection in Pulmonary Tuberculosis: Results and Follow-up of 600 Cases, in press.
10. Longacre, J., Carter, B., and Quill, L.: An Experimental Study of Some of the Physiologic Changes Following Total Pneumonectomy, J. THORACIC SURG. **6**: 237, 1937.
11. Carter, B., Longacre, J., and Quill, L.: A Study of the Changes in Cardiorespiratory Physiology Following Total Pneumonectomy in Young Developing Animals, J. THORACIC SURG. **7**: 326, 1938.
12. Longacre, J., and Johansmann, R.: An Experimental Study of the Fate of the Remaining Lung Following Total Pneumonectomy, J. THORACIC SURG. **10**: 131, 1940.

13. Bremer, J. L.: The Fate of the Remaining Lung Tissue after Lobectomy or Pneumonectomy, J. THORACIC SURG. **6**: 336, 1937.
14. Lester, C., Cournand, A., and Riley, R.: Pulmonary Function of Pneumonectomy in Children, J. THORACIC SURG. **11**: 529, 1942.
15. Cournand, A., Himmelstein, A., Riley, R., and Lester, C.: A Follow-up Study of the Cardiopulmonary Function in Four Young Individuals after Pneumonectomy, J. THORACIC SURG. **16**: 30, 1947.
16. Cournand, A., Riley, R., Himmelstein, A., and Austrian, R.: Pulmonary Circulation and Alveolar Ventilation—Perfusion Relationships after Pneumonectomy, J. THORACIC SURG. **19**: 80, 1950.
17. Birath, G., Crafoord, C., and Rudström, P.: Pulmonary Functions after Pneumonectomy and Lobectomy, J. THORACIC SURG. **16**: 492, 1947.
18. Gaensler, E., and Strieder, J.: Progressive Changes in Pulmonary Function after Pneumonectomy, J. THORACIC SURG. **22**: 1, 1951.
19. Mathey, J., Oustrières, G., and Godeau, R.: La Cavité restante après pneumonectomie, Le Poumon **8**: 273, 1952.
20. Anthony, A. J.: Funktionsprüfung der Atmung, Leipzig, 1937, Ausg. J. A. Barth, p. 106.
21. Baldwin, E. de F., Cournand, A., and Richards, D. W., Jr.: Physiological Classification, Clinical Methods and Analysis, Standard Values in Normal Subjects, Medicine **27**: 243, 1948.
22. Greifenstein, F. E., King, R. M., Latch, S. S., and Comroe, J. H., Jr.: Pulmonary Function Studies in Healthy Men and Women 50 Years and Older, J. Applied Physiol. **4**: 641, 1952.
23. Gilson, J. C., and Hugh-Jones, P.: The Measurement of the Total Lung Volume and Breathing Capacity, Clin. Sc. **7**: 185, 1949.
24. Van Veen, G., Orie, N. G. M., and Hirdes, J. J.: Spirometric Lung Function Investigations. I. A Rapid Constant Volume Method for the Determination of the Functional Residual Air, Acta tuberc. scandinav. **26**: 251, 1952.
25. Bosch, M. W.: The Determination of the Functional Residual Volume (Gas Exchange Between the Lungs and a Closed System Upon Rebreathing. The Calibration of the System), Acta physiol. et pharmacol. neerl. **3**: 184, 1954.
26. Christie, R.: The Lung Volume and Its Subdivisions: Methods of Measurement, J. Clin. Invest. **11**: 1099, 1932.
27. Noyons, A. K. M.: Méthode d'enregistrement continu de la teneur en CO_2 et O_2 des gaz respiratoires au moyen du diaféromètre thermique, servant à l'étude de métabolisme des tissues des animaux et de l'homme, Ann. de physiol. **13**: 910, 1937.
28. Hirdes, J. J., and van Veen, G.: Spirometric Lung Function Investigations. II. The Form of the Expiration Curve Under Normal and Pathological Conditions, Acta tuberc. scandinav. **26**: 264, 1952.
29. Tiffeneau, R., and Drutel, P.: Études des facteurs alvéolaires et bronchiques de la ventilation pulmonaire, J. franç. méd. chir. thor. **3**: 401, 1949.
30. Gaensler, E.: Analysis of the Ventilatory Defect by Timed Capacity Measurements, Am. Rev. Tuberc. **64**: 256, 1951.
31. Van Veen, G., and Hirdes, J. J.: Spirometric Lung Function Investigations. III. The Correlation Between the Magnitude of the Residual Air and the Usable Part of the Vital Capacity, Acta tuberc. scandinav. **26**: 279, 1952.

Erratum Requested by Dr. Robert P. Glover

In the article entitled "An Analysis of Fifty Patients Treated by Mitral Commissurotomy Five or More Years Ago," by Drs. Glover, O'Neill, and Janton of Philadelphia, which appeared in the October issue of the JOURNAL, page 436, it is noted that a number of the patients included were only four and one-half years postoperative at the time of publication so that the cases reported have actually been followed for four and one-half to six and one-half years.

Announcements

THE AMERICAN ASSOCIATION FOR THORACIC SURGERY

Notice of Annual Meeting

The Thirty-sixth Annual Meeting of the American Association for Thoracic Surgery will be held May 7, 8 and 9, 1956, in Miami Beach, Florida. Headquarters will be at the Hotel Fontainebleau.

Requests for Hotel Reservations

These should be mailed directly to Mr. Jack Slone, Director of Sales, Hotel Fontainebleau, Miami Beach, Florida. Please mention this Association, the type of accommodation desired, the date and approximate hour of arrival and departure. If accommodations are desired elsewhere in Miami Beach, please communicate directly with the hotel of your choice.

Thoracic Surgical Forum

The Thoracic Surgical Forum will be continued as a special session at the 1956 meeting. The Forum is primarily for the presentation of short papers on current thoracic research, experimental problems, anatomical studies and modified or new surgical techniques. The inclusion of any considerable amount of clinical material in these reports makes the choice more difficult and the Program Committee reserves the right to reject such papers, or to suggest their transferral to the Regular Program.

Papers at this session will be limited to ten minutes, including the showing of lantern slides or other illustrative material. At the discretion of the President, three minutes may be allowed as the maximum discussion for each paper.

All Thoracic Surgical Forum papers will be published together in one issue of the JOURNAL. Maximum length: 3000 words without illustrations. If illustrated, an appropriate number of words must be subtracted for each cut used.

Abstracts for Papers

Abstracts for the presentation of all papers at the 1956 meeting must be received on or before Dec. 15, 1955, otherwise they will not be considered by the Program Committee.

Abstracts should be labeled "For Thoracic Surgical Forum," or "For Regular Program." The abstracts should contain from 200 to 250 words and accurately reflect the contents of the completed paper.

Five, repeat, five copies of *each* abstract must be sent to the Secretary of the Association, Dr. Paul C. Samson, 3959 Happy Valley Road, Lafayette, California. They should not be sent to the Editor.

Those on the program are reminded that papers presented at the meeting *must* be handed to the secretary immediately after their presentation. If they are not ready at that time there is a chance that they will not be published in the JOURNAL OF THORACIC SURGERY.

Motion Picture Session

Provisions have been made to show a limited number of motion pictures to be selected on a competitive basis. *The films themselves,* together with descriptive material concerning length of presentation, etc., must be received by Dr. Julian Johnson, University Hospital, 3400 Spruce St., Philadelphia 4, Pa., before Dec. 15, 1955.

(Continued on next page.)

It is planned to run the motion pictures in a smaller room at the same time as the scientific sessions. If the films are well labeled, it will not be necessary for the author to be present for comment.

Applications for Membership

Applications for Associate Membership in the Assocation must be received by the Membership Committee not later than Dec. 15, 1955, otherwise, the applications will be deferred for consideration until the 1957 meeting.

Applicants must be sponsored by three Active or Senior Members of the Association. In addition to the signatures on the application form, the sponsors will forward a *separate* letter concerning the applicant directly to:

Dr. O. T. Clagett
Chairman of the Membership Committee
Mayo Clinic
Rochester, Minn.

Sponsors are reminded that *new* letters of recommendation must be forwarded to the Membership Committee should their applicants be held over for reconsideration a second year.

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